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SATURDAY FEBRUARY 6 1943

TABLE OF CONTENTS

Treatment of Cerebral Contusion LAMBERT ROGERS, F.R.C.S. 151

Nature of the Rheumatic Child—II DOUGLAS HUBBLE, M.D. 154

Macrocytic Anaemia of Pregnancy and the Puerperium H. W. FULLERTON, M.D. ... 158

Economy in Fats and Oils in Treatment of Skin Diseases G. H. PERCIVAL, M.D. 160

The Bread Factor in a Wartime Dietary MARION B. RICHARDS, D.Sc. 161

MEDICAL MEMORANDA

Anaesthesia for Laryngofissure. C. B. LEWIS, M.R.C.S., D.A. 162

Combined Infection of the Throat with Diphtheria and Tuberculosis. J. J. LINEHAN, M.D. 162

GENERAL ARTICLES AND NEWS

Help for the Shipwrecked: Report of M.R.C. Committee 168

THE SERVICES 176

UNIVERSITIES AND COLLEGES 176

MEDICAL NOTES IN PARLIAMENT 176

MEDICAL NEWS 178

LETTERS AND ANSWERS

Income Tax—Nasal Defence—*B.P.* Amendments—The Chemist's Shop in Denmark 178

OBITUARY

Sir StClair Thomson, M.D., F.R.C.P., F.R.C.S. 173

Sir Robert Armstrong-Jones, M.D., F.R.C.P., F.R.C.S. 175

Robert Craik, M.D. 175

Epidemiology Section 177

LEADING ARTICLES

New Era in Industrial Medicine 164

Cirrhosis of the Liver 165

ANNOTATIONS

Pathogenesis of Peptic Ulcer 165

Starvation in Southern France 166

Treatment of Diabetic Coma 166

Help for the Shipwrecked 167

B.C.G. Vaccination 167

REVIEWS

Nasal Sinuses 163

A Fifty-year-old Textbook 163

Human Control of Climate 163

Notes on Books 163

SUPPLEMENT

Beveridge Report: A Plan for Social Security 17

Insurance Acts Committee 21

BEQUESTS FOR MEDICAL CHARITY 21

FEES FOR N.D.F.S. MEMBERS 21

INCREASE IN FEES 21

CORRESPONDENCE 22

H.M. FORCES APPOINTMENTS 22

CORRESPONDENCE

Surgery of Oesophageal Cancer. G. GREY TURNER, F.R.C.S. 169

Filaria Worm in Eye. C. R. DUNCAN LEEDS F.R.C.S.Ed. 169

Orbicularis Oris in Facial Paralysis. Sir ROBERT STANTON WOODS, M.D. 170

Phosphorus Poisoning from German Bullets. A. J. BLAXLAND, F.R.C.S. 170

German Explosive Bullets. NORMAN P. HENDERSON, M.B.; C. M. BEADNELL, M.R.C.S. 170

Pasteurization of Milk. J. R. CHARLES, M.D. 171

Diphtheria during Immunization. F. LAWRENCE SMITH, M.B.; D. F. JOHNSTONE, M.R.C.S. 171

Treatment of Ischaemic Contracture. W. GRANT WAUGH, F.R.C.S.Ed. 172

Mitral Systolic Murmurs. C. H. Ross CARMICHAEL, M.B. 172

Mass Miniature Radiography Units. PAUL CAVE, M.B. 172

Ligature of Innominate Artery. Sir W. I. DE C. WHEELER, F.R.C.S.I. 173

Artificial Respiration. W. J. BURNS SELKIRK, M.D. 173

Teaching First Aid. ARTHUR S. G. BELL, M.R.C.S. 173

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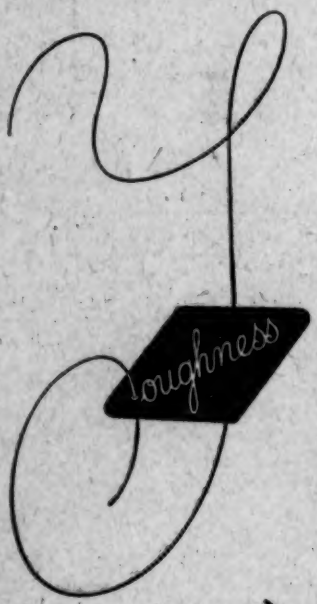

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BRITISH MEDICAL JOURNAL

LONDON SATURDAY FEBRUARY 6 1943

THE TREATMENT OF CEREBRAL CONTUSION*

BY

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Cases of cerebral contusion following so-called "closed" head injuries are usually admitted to beds in the general surgical wards of a hospital, and two tendencies may be noticed in regard to them: first, like fracture cases in the same wards, they are not infrequently regarded rather as necessary evils and relegated to the care of the house-surgeon; secondly, treatment is usually of a symptomatic character—e.g., for restlessness—without due consideration for the complex pathological processes which result from the injury to the brain and are involved in its recovery. It is not surprising, therefore, to find these cases either just left recumbent, or else subjected to various methods of treatment, which for the most part may be described as of an expectant character, consisting merely of rest in bed and efforts to palliate complications as they appear.

What, then, should we do for a patient rendered unconscious by a severe head injury and who may or may not present an obvious lesion such as a scalp wound, for which local treatment is required? Although such cases are of frequent occurrence it is true to say of them that, first because of the extreme variability in the severity of head injuries and of their effects, rarely, if ever, do any two have identical cerebral lesions; and, secondly, even after a prolonged period of unconsciousness some of them make remarkable recoveries, even if given little or no treatment at all—e.g., just left to lie on a bunk, as happened in sailing-ships in olden days. Obvious difficulties, therefore, are encountered in testing the efficacy of any particular form of treatment for such cases. The fact remains, however, that many patients who recover from the immediate effects of closed head injuries do present sequelae in the form of the so-called post-concussional state. We may accordingly ask whether it is not possible to lessen the incidence or altogether avoid this post-concussional state by adopting prophylactic measures in the form of early treatment. It is my belief that it *is* possible, and the object of this paper is to record experiences with a method of treatment which I contend is indicated on theoretical, experimental, and clinical grounds, and which appears to promote the recovery of these patients, with a singular freedom from complications or after-effects.

If, following a particular form of treatment, convalescence is smoother than it is otherwise known to be, and if there is a consistent absence of sequelae known to occur from time to time in cases not so treated, it is suggested that we have a strong argument in favour of that treatment—provided always, of course, that the treatment itself does not add to the mortality of the condition.

Testing the Value of Treatment

What methods have we for testing the value of a particular form of treatment? The answer is not as simple as it might seem to be at first sight. It would appear that all we had to do was carry out direct experiment—i.e., test the remedy; but it has already been pointed out that many patients recover whether a particular form of treatment is given or not, and they may do so even despite it. Next, since no two cases of head injury—which term implies a complex and variable pathology—are quite alike, it is difficult to obtain controls by which to effect anything like exact comparison. Lastly, while a certain amount of information is to be gained from animal experiments, there are obvious fallacies in transferring the results of such experiments to man or in attempting to draw conclusions from clinical observations made on animals. To test the evidence, therefore, the most scientific course will be to adopt a combination of methods such as the following:

(i) To examine the argument for the particular remedy by careful consideration of the premisses on which it is based and the conclusions to be derived therefrom.

(ii) To review the experimental evidence bearing on the argument.

(iii) To examine the results of a series of treated cases, followed over some years, particularly noting the incidence of complications known to occur from time to time with other methods of treatment.

(iv) To review a few cases in some detail while, so far as possible, making allowance for the qualifications already mentioned.

It is first necessary to indicate the type of treatment. Briefly, this consists of continued gentle dehydration, instituted with the object of withdrawing cerebrospinal fluid from the intracranial cavity and so providing potential space into which brain-swelling can take place, thereby avoiding compression of its blood vessels.

I. The Argument

The argument in favour of this form of treatment is briefly the following: Bruised tissue, whether that comprising the brain or any other organ or structure, must be permitted to swell if it is to recover completely. If by confinement within a limited space swelling is prevented there must be a reduction in blood supply owing to the compression of the vessels supplying the damaged tissue, and this reduction is detrimental to complete recovery.

While to-day the Monroe-Kellie doctrine may not be wholly acceptable in the form in which it was originally propounded, it is probably true to say that with certain reservations the data on which it is founded are true and that in general it holds good. There is a relatively fixed total volume of the cranial contents, but with a capacity for change in any one of the three chief elements comprising it—viz., the brain itself, the circulating blood, and the cerebrospinal fluid. The most practical method of providing for swelling of the bruised brain is to reduce the amount of the cerebrospinal fluid within the skull. This may be conveniently done by dehydration.

If tissue-swelling is allowed to take place after injury the subsequent recovery of the tissue is striking; if, however, swelling is restricted or prevented altogether, a varying degree of permanent damage follows. This important principle of pathology is exemplified when an organ readily capable of expansion—like the kidney—is compared in its reaction to injury with an organ restricted by its capsule from expanding—like the testis. The renal capsule is thin and readily distensible; the testicular capsule (tunica albuginea) is tough, fibrous, and comparatively inelastic. The bruised kidney swells, recovers, and functions normally; the bruised testis cannot swell, and subsequently atrophies. The cerebral envelopes are even more

* Based on a lecture delivered at the Royal Naval Hospital, Plymouth, Jan. 7, 1943.

rigid than the tunica albuginea, and the only way in which some variation can be given to their limits of constriction, other than by craniotomy (with its attendant risks and disadvantages), is by reducing the volume of the cerebrospinal fluid surrounding the brain, the fluid itself being incompressible. As Wilfred Trotter (1929) has pointed out, a simple bruise, of no ultimate importance to an organ with a yielding capsule, is a serious matter with the brain, which must be allowed to swell if its circulation is not to be materially interfered with. If this swelling, which is the normal reaction to injury, is restricted, the blood supply to the bruised region is diminished, and the ensuing ischaemia may well result in cortical atrophy with associated ventricular distension or become manifest by symptoms such as those which comprise the post-concussional state. This post-concussional state may conceivably represent incompletely resolved cerebral contusion or subsequent atrophy consequent upon failure to provide for brain-swelling following injury.

Brain-swelling.—In the case of the brain, injury produces swelling very readily and to an extreme degree. Thus, Dandy (1941) writes: "Trauma that would cause no concern to other hardier tissue produces severe swelling of the brain." Some idea of the extent of this swelling and the rapidity with which it occurs can be gathered from cases of cerebral tumour which have undergone operative treatment. In this connexion the same author writes: "The large cavity created by the tumour's removal may be entirely obliterated in less than half an hour, and with no gross haemorrhage."

One of the arguments advanced against the routine use of dehydration to permit brain-swelling to occur in cases of closed head injuries is that from time to time the pressure of the cerebrospinal fluid in the lumbar pond of patients unconscious as a result of a severe head injury has been found to be low. This, however, does not invalidate the thesis or contraindicate dehydration—first, because experimentally (White *et al.*, 1942) no correlation has been found to exist between cerebrospinal fluid pressures and the degree of cerebral swelling (e.g., as produced during anoxia); and, secondly, because variations in the pressure of the fluid in the lumbar pond take place so frequently that an isolated observation that at one particular time it is low is probably of little significance. Finally, it is reasonable to assume that such pressure differences as occur during brain-swelling are chiefly of a local character and may require intracellular observations to detect them; or, as White (1942) has recently suggested, in head injuries there is often concomitant anoxia of the brain due to depression of the respiratory centre, and this anoxia may considerably reduce the secretory activity of the choroid plexus.

Still further evidence that the findings at lumbar puncture are not necessarily an indication of the intracranial fluid state has been found at operation; thus Bathe Rawling (1918) wrote in connexion with subtemporal decompression which he was at that time performing for cases of suspected cerebral oedema: "In one of my last cases four punctures carried out immediately previous to operation showed a great diminution in the amount of cerebrospinal fluid; and, in the operation performed immediately afterwards, the opening of the dura showed a most typical condition of cerebral oedema with very great excess of fluid."

II. Experimental Evidence

Experimentally it should be possible to ascertain whether brain-swelling takes place following head injury: White (1942) has found a very constant swelling of the brain of cats, even after the animals have had mild concussion—e.g., the brain, instead of being 10 to 11% smaller than the intracranial chamber, was found to be only 7 to 8% smaller. Surgeons (including myself) who have operated on cases of closed head injury within the first few hours have commented on the swollen condition of the brain. Furthermore, it is a widely held view, and probably true, that when a compound fracture with its immediate decompressive effect has occurred convalescence is easier and freer from complications than when a comparable type of injury has produced cerebral contusion alone.

Turning next to the other side of the problem, Weed and McKibben (1919) constantly found histological changes in the brains of animals given intravenous injections of hypertonic or hypotonic solutions if by keeping the skull closed the brain volume was not allowed to alter. On the contrary, however, when the brain volume was allowed to alter by opening the skull with a trephine these histological changes did not take place.

Provision for Brain-swelling.—If brain-swelling which is the normal reaction to injury is allowed to take place the blood supply remains relatively unimpaired and resolution and recovery are complete. If the swelling is restricted local blood supply is impeded and may be followed by changes which are evident clinically as the "post-concussional state." Theoretically, provision may be made for brain-swelling by performing craniotomy or by withdrawing the incompressible cerebrospinal fluid from the skull; for, as Dandy (1933) has pointed out, "space compensation is the principal function of the cerebrospinal fluid." This fluid-withdrawal may be either by lumbar punctures or by dehydration, or it may be effected by a combination of both methods. In practice it has been found that craniotomy is unnecessary and undesirable and that dehydration is the procedure of choice, probably because this is a continuous and gentle process, whereas repeated lumbar punctures result in a rapid to-and-fro swing of cerebrospinal fluid production and thus are only occasionally employed to augment dehydration or for diagnostic reasons.

Dehydration by Magnesium Sulphate.—The question arises whether it is possible by any simple measures to produce the continued gentle dehydration previously advocated, and the reply is that this can be effected by instillations of magnesium sulphate into the rectum at regular intervals. Cushing and Foley (1920) showed that 20 to 30 c.cm. of a saturated solution of sodium chloride introduced into the duodenum or the rectum of the cat produced a maximal fall of cerebrospinal fluid pressure precisely comparable to that which occurred when the solution was given intravenously. They pointed out, moreover, that when the salt is given by the alimentary route the intracranial changes which are produced are not only not attended by disturbances of pulse, respiration, or blood pressure, but that possibly, also, alteration of the cellular elements of the blood is avoided. Temple Fay (1924) found that magnesium sulphate instilled into the rectum had advantages over sodium chloride in causing less discomfort and being almost twice as efficient in its effects on the cerebrospinal fluid. Being non-dialysable and therefore not absorbed into the blood stream, magnesium sulphate does not produce a secondary wave of tissue oedema with an increase in intracranial pressure as does the sodium salt. Writing of the rectal use of magnesium sulphate in the University Hospital in Philadelphia, he states: "The beneficial use of this salt in cranial injuries has been well established in this clinic." This has also been our experience at Cardiff. Its use in the treatment of cerebral oedema has also been endorsed by Donald Munro (1938) and others.

The details of treatment are briefly as follows: (1) The patient is propped up in bed with the head raised to reduce intracranial venous congestion, and for the same reason it is necessary to make sure there is no constriction of the neck veins by tight pyjama collars or other garments. (2) Retention enemas of 6 oz. of 30% magnesium sulphate solution in water are given every six hours. These must be very gently and slowly administered so as to be retained. (3) Fluid intake by mouth is restricted and a certain degree of thirst is a criterion of effective dehydration.

III. Examination of Results

In 1929* this practice was begun in the surgical unit at the Cardiff Royal Infirmary, and has consistently been followed since in all cases of head injury severe enough to be admitted (Lambert Rogers, 1935, 1937, 1938a, 1938b). The majority of these patients were in a state of profound or prolonged unconsciousness. In the years between 1929 and 1940 550 cases were treated. Fractures of the skull were detected in 133 of these, but there is reason to believe that many others had basal fractures not revealed by *x*-ray examination. Of these patients 25 died, the majority soon after admission.

An absence of both immediate and late complications has been noticed. Traumatic delirium (cerebral irritation) was

* The idea followed Wilfred Trotter's teaching of the pathology of brain injury, and I had thought we were the first to institute the practice, but have since read that this treatment was in operation in 1921 on the neurosurgical service of the late Dr. Charles H. Frazier (Temple Fay, 1935).

infrequent, and when it did occur was only of a mild character. The absence of the noisy, shouting, restless type of patient with head injury, previously so often seen, was a welcome change which has often been commented upon by those taking part in the dehydration treatment. Furthermore, if focal signs arose in the course of treatment these were considered to indicate craniotomy, and were found at operation to be caused by blood-clot and not oedema, which in some cases previous to the dehydration regime had been revealed when these cases were operated upon because of the onset of focal signs. Lastly, although a follow-up clinic is maintained in connexion with the surgical unit, not one of these 525 cases has required readmission because of the so-called post-concussional state or sequelae of any kind. It is tempting to doubt, therefore, whether there is such a condition as the post-concussional state if brain-swelling is permitted to take place at the time of injury, and whether in fact this state, as has already been suggested, does not represent incomplete resolution the result of failure to institute effective initial treatment. It is perhaps pertinent to remark that during the period under consideration cases of so-called post-concussional state were admitted from other clinics where dehydration had not been carried out.

IV. Review of Some Cases

The following details of a few cases exemplify the result of this treatment.

Case 1 : Fracture of base of middle fossa extending into right parieto-occipital region of vault. Cerebral contusion and laceration. Spastic paraplegia, right brachial monoplegia, left facial palsy, cyanosis, coma three weeks, cerebral irritation, bilateral suppurative otitis. Dehydration 15 weeks. Recovery complete.—A physician aged 53 was admitted after a motor accident, a fast-travelling lorry having collided with the side of the taxi-cab in the back seat of which he was sitting. On admission he was cyanosed and in profound coma; respirations were deep and noisy; he was bleeding from both ears and nostrils. There was a superficial scalp wound on the forehead and a haematoma over the occiput. There was a slight right facial palsy; the right arm was spastic, the left flaccid; there was spasticity of both legs, and the plantar reflexes were extensor. Death appeared to be imminent. He was propped up and dehydration begun. Lumbar puncture two days after admission showed a pressure of only 100 mm. water and red opalescent fluid; 20 c.cm. were withdrawn, and this was repeated 2 days later. Systolic blood pressure, 164 mm. Hg. Radiographs showed a "vertical fissure in the right occipito-parietal region." At this time he was groaning unceasingly, and was rather restless and still profoundly unconscious; the right arm was paralysed, but he was moving his left arm and both legs. The facial palsy had increased slightly. Three weeks later he developed suppurative otitis media in both ears. He was irrational but recovering consciousness, though unable to recognize an old friend. Later still the plantar reflexes became flexor; he was less excitable and had no headache, but complained of tingling in his right arm. Blood pressure, 130 mm. Hg. Dehydration was continued for a period of 15 weeks. Recovery was complete, and he returned to active consulting practice. For the seven years which have since elapsed he has worked consistently, is apparently normal in all respects, and is at present working harder than ever.

Case 2 : Linear closed fracture. Cerebral contusion, coma, delirium, right monoplegia soon proceeding to hemiplegia, middle meningeal haemorrhage. Bone flap; removal of 83 g. of extradural blood-clot. Dehydration 1 week. Recovery complete.—A farmer aged 33 was involved in a brawl in a local inn and hit with a ploughshare on the left side of his head. He was a big muscular man, over 6 ft. tall, and on admission was unconscious and very restless. Pupils reacted very sluggishly to light and both plantar reflexes were extensor. Dehydration was begun on admission. He remained unconscious for three days, but gradually became more docile as he was dehydrated. He then developed a right monoplegia which soon progressed to a hemiplegia. The systolic blood pressure was 90 mm. Hg. A linear fracture traversed the left parietal and squamous temporal bones. A left parietal bone flap was turned down and 83 g. of extradural blood-clot removed. He made a complete recovery, power returning first in the leg and then in the arm.

Case 3 : Closed head injury—probable basal fracture—unconscious 48 hours. No dehydration; intense headache, bradycardia; semi-conscious and very restless when seen. Dehydration 14 days. Rapid improvement and recovery.—An Army officer aged 35 fell from a lorry, struck his head, and was admitted unconscious to a cottage hospital. He was kept lying in bed for five days, and when then seen was semi-conscious, restless, vomiting, and complaining of intense headache. The pulse rate was 56; the cerebrospinal fluid was intimately mixed with blood. He was propped up and dehydration begun. The change was rapid, his headache being relieved, the vomiting ceasing, and restlessness subsiding. The matron of the cottage hospital volunteered the remark that the magnesium sulphate instillations relieved his headache. He was dehydrated for a fortnight, and was discharged symptom-free.

Case 4: Closed fracture, right parietal, following fall of 20 ft. Unconscious 2 hours, subsequently drowsy. Dehydration 5 days. Recovery complete.—A dockyard labourer aged 22 had an epileptic fit and fell from some staging about 20 ft. He was admitted to hospital, remained unconscious two hours, and was subsequently drowsy. He remembered feeling he was going to have a fit and then later finding himself in hospital. He was dehydrated for five days. The medical officer reported that he became less drowsy after the dehydration and made a complete recovery.

Case 5 : Closed fissured fracture, left parietal, following fall from lorry. Unconscious for few minutes. Wound of pinna. Dehydration 3 days. Recovery complete.—A marine aged 35 fell out of the back of a lorry and was unconscious for a few minutes only. On admission to hospital he had headache, was drowsy, and desired only to sleep; his cerebration was slow. He was dehydrated for 3 days, and the medical officer noted: "Patient benefited from dehydration. It relieved headache and he became brighter."

Case 6 : ? Fracture in parieto-occipital region. Closed head injury. Fall off back of lorry; unconscious 15 hours. Intense headache. Dehydration 7 days. Recovery complete.—A pensioner yeoman of signals aged 42 fell off the back of a lorry and was admitted unconscious. He remained so for 15 hours, and, although when he regained consciousness he was mentally alert, he was depressed and had intense headache. Medical officer noted: "Patient obtained benefit from dehydration. Headaches were relieved."

Case 7: Closed injury. Patient found lying unconscious beside bicycle in road. Abrasions of face, hands, and leg. Dehydration 18 days. Recovery complete.—An A.T.S. officer aged 30 was admitted unconscious to hospital, having been found in the road beside her bicycle. She remained unconscious for 6 days and was then unresponsive and drowsy, and complained of intense headache. The optic disks showed slight papilloedema, and the pulse was consistently below 60. The electro-encephalogram showed no abnormality in any area, but she was not made to overbreathe. When she was first seen either brain-swelling or a subdural haematoma was suspected. Remarkable changes followed dehydration. Once this was effectively instituted she made an uninterrupted and rapid recovery, and was discharged symptom-free.

Case 8 : Closed injury following fall off lorry; semi-conscious, traumatic delirium, hemiplegia. Dehydration 3 weeks. Recovery.—A soldier aged 25 fell out of a lorry on to the road and was admitted to a local hospital in a semi-conscious condition. He became very restless and irritable, and was noticed to have a hemiplegia. When seen 48 hours afterwards he was still only semi-conscious, very restless, irrational, irritable, and hemiplegic. The right plantar reflex was extensor, the pulse between 50 and 60. Lumbar puncture showed blood intimately mixed with the cerebrospinal fluid. Following dehydration he made a rapid recovery, his symptoms disappearing from the time of its institution.

Not once, but many times recently, I have had the experience of finding a young Service patient with a head injury lying flat in bed, unconscious, irritable, and restless, and within a few hours of propping him up, doing a lumbar puncture, and beginning dehydration therapy, seen him become a conscious rational being once more. I attribute such changes to the recognition of the importance of brain-swelling and the provision thus made for it.

While there have been difficulties in getting this treatment accepted as a routine measure in this country and few British neurosurgeons, with the exception of Wylie McKissock (1942), have openly supported it, there have not been lacking elsewhere those who have upheld the claims made for it. In the United States Foster Kennedy and Wortis (1932) have advocated dehydration treatment, and Temple Fay (1937) claims not only a reduction in mortality of somewhere about 25% in head injury cases so treated but that there is a remarkable improvement from the standpoint of subsequent economical and social adjustments. He writes: "If its [the brain's] tissue can be maintained alive during the crisis, a prompt return of function will follow to a far greater degree than has formerly been recognized. . . . What surgeon would permit a patient with a bed-sore to continue pressure on the area and expect the devitalized anaemic tissue to recover its function? Yet many . . . permit a brain to become involved in a relentless hydraulic compression, squeezing the blood out of this vital and important tissue." On the Continent, Decker (1938) of Lausanne has upheld its value, claiming a lowering of mortality and improvement in results since its adoption, and there have been others who from time to time have spoken or written in favour of it.

But for the most part these have been "voices crying in the wilderness." Anything like routine appears to be abhorrent to some surgeons, yet the reaction to injury must at least be always in the same direction, and if bruised brain always reacts by swelling, treatment surely should always be directed to providing for the swelling to take place.

Conclusion

To sum up, then, we may inquire whether the Monroe-Kellie doctrine does hold in regard to the reaction of the bruised brain to injury. Is the encapsulation of the brain by cranium, meninges, and cerebrospinal fluid of a comparatively rigid and inelastic character like the tunica albuginea of the testis, as has already been suggested, or does it by some physiological mechanism readily accommodate to cerebral swelling as does the thin elastic renal capsule to swelling of the bruised kidney? If, as it seems to me, the encapsulation behaves in a comparatively rigid and relatively inelastic manner, there can be no questioning the desirability of directing our treatment of the bruised brain towards an attempt to render the cerebral encapsulation more accommodating and thus to maintain the blood supply to the contused area by permitting it to swell in response to the law of its reaction to injury. It is further contended that the best method of doing this is by continued gentle dehydration to facilitate brain-swelling and so prevent the reduction in the blood supply to the damaged areas of the contused brain.

There is some reason to think that our forefathers may have had some inkling of the benefits of dehydration. Ambroise Paré over 300 years ago advocated restriction of fluids, purgation, and sweating for cases of head injury (Turnbull, 1939); and the prescribing of calomel and salts and otherwise purging of patients with fracture of the base of the skull, which was common practice early in this century, was probably introduced quite empirically, but was nevertheless essentially sound.

Perhaps in addition to the striking clinical improvement which is so often observed as soon as this treatment is instituted, the absence of sequelae in a long series of cases is the best criterion on which to base its claims. It is simple—so simple that it tends to be neglected or perhaps rather scornfully regarded, as Naaman was inclined to treat the advice of Elisha (ii Kings v). The Syrian captain was angered and disappointed that the prophet had not come down and performed some miracle. In these cases of severe closed head injury with profound unconsciousness it would be so much more dramatic to perform wide decompressive craniotomy, but the simpler treatment is so effective that, like bathing in the Jordan, it suffices. To those who are still not convinced by the arguments here put forward, the figures presented, or the cases instanced, one may quote what was once graphically said of some numerical evidence: "An emaciated child suffering from hunger is a more impressive sight than any number of mortality statistics." A few cases treated by this method will prove more convincing than any theoretical considerations.

Summary

Prolonged unconsciousness following a severe head injury, particularly when blood is intimately mixed with the cerebrospinal fluid, would indicate that the brain has been bruised or torn. Evidence is presented that the brain reacts to such injury by attempting to increase its volume.

No two head injuries are exactly comparable, and it is well known that remarkable and complete recoveries are often made from extensive injuries to the skull and brain. It is difficult, therefore, to assess the value of any particular type of treatment. The absence of sequelae following the consistent application of any particular form of treatment, however, may be urged in its favour.

Dehydration is a method of permitting brain-swelling to take place after injury, and the evidence in favour of its application is here examined. It should be effected by rectal magnesium sulphate and not by intravenous methods, which probably produce undesirable reactions.

Smoothness of convalescence and an absence of sequelae—e.g., the so-called post-concussional state—have been noticed after treatment of a large series of cases by continued gentle dehydration.

It is suggested that the post-concussional state may, in many cases at least, be the result of failure to introduce effective treatment in the early stages and represent a condition of unresolved contusion, it being maintained that resolution takes place readily and completely if the requisite condition for reparation is provided. It is suggested that this is the provision of space so that brain-swelling can occur and is not restricted by the cerebrospinal envelopes, with consequent impairment of the cerebral blood supply. Provision for such swelling is made by sustained dehydration, which reduces the incompressible water content of the skull.

References

Cushing, H., and Foley, F. E. B. (1920). *Proc. Soc. exp. Biol.*, N.Y., **17**, 217.
Dandy, W. E. (1933). *J. Amer. med. Ass.*, **101**, 772.
—— (1941). Dean Lewis's *System of Surgery*, vol. **12**.
Decker, P. (1938). *Helv. med. Acta*, **5**, 808.
Fay, Temple (1924). *J. Amer. med. Ass.*, **82**, 766.
—— (1935). *Ann. Surg.*, **101**, 76.
—— (1937). *Surg. Clin. North Amer.* **17**, 1661.
Kennedy, Foster, and Wortis, S. B. (1932). *J. Amer. med. Ass.*, **98**, 1352.
McKissock, Wylie (1942). *Proc. roy. Soc. Med.*, **35**, 757.
Munro, D. (1938). *Cranio-cerebral Injuries*, New York.
Rawling, L. Bathe (1918). *British Medical Journal*, **1**, 499.
Rogers, Lambert (1935). *J. roy. nav. med. Service*, **21**, 2.
—— (1937). *J. Int. Chir.*, **3**, 109.
—— (1938a). *British Medical Journal*, **1**, 685.
—— (1938b). Ibid., **1**, 743.
Trotter, W. (1929). *Nature*, **123**, 533.
Turnbull, F. (1939). *Canad. med. Ass. J.*, **40**, 579.
Weed, L. H., and McKibben, P. S. (1919). *Amer. J. Physiol.*, **48**, 550.
White, J. C. Personal communication, 25/8/42.
—— Verlot, M., Silverstone, B., and Beecher, H. K. (1942). *Arch. Surg.*, **44**, 1

THE NATURE OF THE RHEUMATIC CHILD

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(*Concluded from p. 125*)

Aetiological Factors in the Rheumatic State

It should be generally conceded that statistical inquiry has a more limited value in such a disorder as the rheumatism of childhood than it has in many other diseases. Diagnosis is more than usually difficult, so that the outlook, the experience, and the bias of the individual clinician have to be assessed. The conclusions, too, are valid only for the time and place in which they are made. This may be true in some degree of all statistical investigations, but it is most true of diseases that are disorders of personality and environment. So it should surprise no one that careful investigations by experienced observers at different times and in different places arrive at contrary conclusions with regard to the aetiological factors of rheumatism. The influences of climate, of heredity, of poverty and its accompaniments, are still keenly debated. It may be said of climate that its influence is equivocal; the disease is certainly not limited to northern latitudes and temperate climates as was once taught (Swift, 1940; Fernando, 1939), and it seems probable that the higher incidence of rheumatism in cold than in hot climates depends on the increased liability to respiratory infections in these areas. In England the geographic distribution of the disease cannot be shown to depend on climatic factors (Morris and Titmuss, 1942).

It is generally admitted that the disease shows a well-marked familial incidence, but in Great Britain most observers agree that this depends more on exposure to similar environmental stresses than on an inherited quality. In America, observers are inclined to attach more importance to the influence of heredity, but there are many facts (some of which are dealt with below) which suggest that the heritable factor cannot be of dominant importance. There are, however, three factors—age, social grouping, and the haemolytic streptococcus—about which opinion may be more dogmatically stated. Of these factors it may be said that unless an individual is between the ages of 3 and 15 it is uncommon for him to develop the disorder for the first time; that if he belongs to the wealthier classes he will with great probability escape it altogether, and that, if he does develop it, then at some phase of the illness, but not always at the start, the presence of the haemolytic streptococcus can be demonstrated. These factors deserve more attention here.

Age

There is no other infection which, while conferring no immunity, limits itself so accurately to an age group. This fact alone should be sufficient to characterize it not as a disease caused by an invader but rather as a disease conditioned by the host. This age group is paramount. Why?

The peculiarity of this age group is growth, and yet children grow more quickly in the cradle and continue to grow after puberty, so that physical growth does not suggest a solution. The fact that chorea is limited to this same age group confirms those who believe that rheumatism is an infection in their belief that chorea too is caused by the rheumatic invader. On the other hand, those who believe that chorea is a profound nervous instability, proved to follow psychic stress, will be encouraged by this age-grouping to look for a comparable nervous instability in non-choreic rheumatism. Physical growth is a silent progress to predestined stature (always granted a guaranteed nutrition). The growth of personality is a pilgrimage attended with difficulty and beset by dangers. The rheumatic age embraces the years when the child steps down from its cot into the family, and then out into the world—the years of integration and adjustment. I take it to be no coincidence that these are the years of growing pains, of chorea, and of rheumatic carditis.

Social Grouping

There is general agreement on both sides of the Atlantic that child rheumatism is much more frequent in the poorer classes. There are no comparable groups of statistics for the children of the poor and the rich; but Still (1927) found it to be nearly 20 times more common among his hospital out-patients than among the children of the same age group in his consulting practice. Glover (1930) suggests that it is 30 times more common among the poor. This is not to suggest that it is an unknown disease among the well-to-do. American writers often emphasize this (Kaiser, 1934), and epidemics among relatively closed communities of wealthier children in this country have been described (Bradley, 1932).

Large-scale investigations in Great Britain made by the Medical Research Council (1927) have established the fact that the incidence of rheumatism is not in direct relation to the degree of poverty.

> "Indeed," writes Still, "if the hospital patients are divided into three classes according to the degree of poverty, the poorest of these three, the very poor and destitute, show a lower incidence of rheumatism than the other two. It would seem, therefore, that if poverty is a factor in the incidence of rheumatism it must be on account of some condition or conditions associated therewith which are not equally present in all degrees of poverty."

Although this conclusion has been contested by some, there should be no doubt that it was valid for the time and place at which the "investigations" quoted were made. Morris and Titmuss (1942) have questioned it on the grounds that the mortality rates show a steep rise in deaths from heart disease coincident with decline in social status right through to the lowest social group. This criticism carries an obvious fallacy, since it might be expected that once the extremely poor child had become rheumatic its chance of survival was less than that of its wealthier brethren; mortality rates from heart disease in social groups cannot be accepted as an adequate reflex of the incidence of rheumatic disease in those groups.

Findlay defines this social status more closely: "It would seem, however, that it is not the very poorest but rather, if one may so express it, the better-class poor families that have been better off and have fallen on evil days." The Medical Research Council report thus described these families, which it labels "Group B," Group A being the less poor and Group C the very poor: "They are normal households, for the most part with adequate furniture, but have no margin for anything beyond the bare necessities of life, and are liable to periods of deprivations should loss of work occur. They have few outside interests, and are mainly occupied with the struggle to make ends meet."

No observer has suggested that the mental and nervous stresses of poverty on children may be as important as the physical. It needs little imagination to see that the nervous stresses of poverty will be greater in Group B than in Group A or C, greater than in the less poor and in the more poor—that is, in the family which has known failure and is pre-occupied with the fear of falling again, with its struggle to keep up appearances, with its avoidance of social life, with its emphasis on hard work as a prelude to success, and with the distortion of its emotional life by fear and insecurity: this is the domestic background to Group B families. Is it fantastic to see in this environment one of the breeding-grounds of child rheumatism? In which class should we expect to find that education imposes a greater nervous strain? Although this factor of nervous instability may explain the higher incidence of rheumatism at this particular economic level of poverty, the general association of rheumatism with poverty at all levels probably depends, as a host of observers have assumed, on the physical accompaniments of poverty, poor clothing, bad and damp housing, malnutrition, and over-crowding. While most of these factors operate by reducing the resistance to infection, others such as overcrowding presumably act by increasing the number and virulence of naso-pharyngeal infections.

Strong evidence against the importance of a heritable factor was provided by the M.R.C. report. Evidence was collected from the Poor Law residential schools, and it was found that children living in these institutions much less frequently developed the rheumatic state than children of the same class living at home. At two of these schools, in 22 and 18 years respectively, there had been only five cases of chorea and five cases of acute rheumatism out of more than 6,000 children who had passed through them. It may be estimated that 200 of these children would during that time have developed rheumatism or chorea had they remained in their own homes. Rheumatism in childhood is a disorder based on environment, although a predisposition may be inherited.

The Haemolytic Streptococcus

There is in any rheumatic child a phase in the development of the rheumatic state which is intimately associated with infection, and this infection can usually be shown to be associated with the haemolytic streptococcus. Sometimes, when the rheumatic state develops three weeks after an acute tonsillitis, the "invader" seems to have sufficient power by its own strength and numbers to overwhelm the body defences; sometimes, as in the subacute rheumatic state and in chorea, there has been preliminary "fifth column activity" in the nervous system which has weakened the capacity for resistance to the invader. There is no means, except by clinical approach, of assessing the relative importance of these various factors in any individual or of estimating their aetiological importance in various peoples. It is certain that these factors will vary in importance with race, with country, with social grouping, and with sex.

The view that emotional disorder may lower the resistance to infections may be difficult for some minds to accept, but it is in fact a commonplace of paediatrics. Cameron (1929) writes of the nervous child:

> "Lowering of resistance to infection of all sorts is clearly incurred. The frequent illnesses of the delicate only child have a real existence. They are not to be explained as imaginary ills due to the too great anxiety of the mother. The timid excitable child, with his faulty physique and wrong posture, not only catches every infection possible but responds to each with a violence peculiarly his own."

This review of the aetiological factors in the rheumatic state suggests that the nearly absolute limitation of that condition to an age group and its common association with poverty do not refute the theory that nervous instability in the child is part of the rheumatic diathesis, nor does the recognized association with infection in some of its phases controvert such a theory.

What Manner of Child is This?

Less than 10% of the children entering the rheumatic state do so by way of subacute rheumatism, and in my own cases these children have more often shown evidence of continued nervous instability than of infection. More than 25% of the children entering the rheumatic state do so by way of chorea, and it is suggested that this condition also is evidence not of infection but of profound nervous instability. If these aetiological assumptions are accepted then a third of the children who develop rheumatic carditis show evidence of preceding nervous instability. If this nervous instability is a

component of the rheumatic state can it be shown to be present in other children with active rheumatism who have not suffered either from subacute rheumatism or from chorea?

I attempted to find evidence of this nervous instability in 21 consecutive cases of active rheumatism, none of these patients having suffered from chorea or from anything resembling subacute rheumatism. I did not regard the history as displaying nervous instability unless the child showed some disorder of physical process such as insomnia, somnambulism, or night terror; anorexia, diarrhoea, or acquired enuresis; fatigue, fidgetiness, or habit-spasm; pains, whether of head, abdomen, or limbs—and in addition some evidence in the mother's story of emotional disorder or environmental difficulty.

The results of this inquiry were disappointing—only three of the 21 children showed evidence of nervous instability when judged by this exacting standard. The parents of two other children, however, told a story of environmental stress that seemed so closely related to the onset of the rheumatic infection as to provide an aetiological factor. Neither of these two children showed any previous psychological abnormality. One other point of importance emerged. The parents of 16 of these children described them as unusually excitable—such excitability, for the most part, going back to the "toddler" age. It was notable, too, that eight of these mothers used the expression "inwardly excitable." Of these 21 children 12 were described by their mothers as unusually timid or shy. It was as a rule easy to detect evidence of excitability or timidity in these children. This inquiry disappointed me until a year ago, when I read an article by Neustatter (1937) which began with a remark made by Prof. H. A. Harris of Cambridge, "The rheumatic child is highly strung but he isn't neurotic." I realized then that I had been "barking up the wrong tree" in looking for evidence of neurosis in these children. The important fact is their excitable temperament, demonstrated again by this small series of mine, but emphasized over many years by a number of clinicians. Cheadle wrote in 1889:

"I would insist on the fact that there is nothing antagonistic between the agency of the nervous state or mental excitement and rheumatism. They are concerned together as factors in many cases of chorea." And again: "I should like to call attention to the relation of nervous excitability to rheumatism. The rheumatic children who develop chorea are intellectual, highly strung, excitable, nervous, often emotional. The question arises, Is the nervous instability due to the influence of the recent active rheumatic state and induced by it; or is the nervous instability part of the original constitution of the child, and chorea produced when this is acted upon by a second fact—viz., rheumatism? Or do the two tendencies run together in the original diathesis—the child with a tendency to rheumatism inheriting thereby a mobile nervous system?"

Still (1912) writes:

"To my mind one of the most striking features in such children is the nervous temperament, to which Goodhart has specially drawn attention. The rheumatic child is *par excellence* the nervous child. In many cases this is seen in an undue excitability: such children will become almost uncontrollable in their excitement over games and amusements or, on the other hand, there may be excessive timidity; in some of the older children this is replaced by a sensitivity or shyness which is almost morbid."

This description of the rheumatic child's temperament has been elaborated by many authors, but the quotation from Still described it well enough—the excessive excitability disguised often by shyness but wildly expressed in moments of acute emotion. It has often been suggested that this nervous excitability is the result of rheumatic infection, but Neustatter's investigations do not support such a theory. He concludes that the "rheumatism may aggravate the nervousness but does not cause it." This has also been my experience. The mothers of these children related that the excitability was present for years before the development of the rheumatic state. Therefore studies of the child's personality during and after the development of the rheumatic state will have equal validity with studies made before; and they should be more informative, since the child's nature has been exposed to the most searching of all tests—a recognition that the heart, the organ whose beating signifies life and whose stillness means death, is diseased. Poynton (1936) said of this emotional disturbance, in the Harben Lectures:

"We must understand the child with acute heart disease. It may be instinctively terrified to a high degree, just as an adult may be when he or she feels that the very pivot of life is in danger."

Winnicott (1938), who has studied these children for many years as a paediatrician and later by psycho-analytical technique, writes:

"When we examine the child's heart we do not always realize that in the child's unconscious fantasy we are testing his ability to keep his ideals alive and unhurt. The child does not necessarily understand the life and death of his body as we understand it, but he understands something deeper which is the life or death of what he believes in and values most in himself. What we say, therefore, about the things inside to which we listen with a stethoscope and discuss with our colleagues over a bedside comes to the child as a matter of life and death in an acutely real sense. It is a matter of reality and of the right to live. In adult terms, it is to be or not to be, to continue to live or to seek death. These are the anxieties that belong to psychiatric depression."

It makes little difference whether we choose to talk of instinctive terror with Poynton or in terms of unconscious fantasy with Winnicott, the important fact for practising physicians to realize is that we are dealing with children who are suffering from fear—unexpressed, unrecognized, and undischarged.

Neustatter suggested that the symptoms—night terrors, restless sleep, worrying disposition, timidity, swings of mood, and emotional instability—which were commoner in his rheumatic groups have an aetiological distinction from the neuroses: "the former are possibly of primarily physiological origin and the latter due to psychological causes." This idea is an illuminating extension of Prof. Harris's remark quoted above, but its expression and the juxtaposition of "physiological" and "psychological" are not happy. It is preferable to regard the temperament of these children as showing a quantitative difference from normal—a speeding-up of both motor and emotional reactions—while the neurotic displays a qualitative deviation from normal. It is possible that the electroencephalogram may one day diagnose and measure for us this quantitative deviation.

No one can deny the emotional and kinetic lability of the child with chorea, but it is as legitimate to deny as it is to assert that this excitability exists also in many rheumatic children. Nor do I see how, in the present state of our knowledge, these matters can be put to scientific proof. There is no laboratory test for acute rheumatism, nor is there any objective measurement of excitability; investigation can only be by question and answer and by subjective observation. Any one physician caring for any one rheumatic child has to attempt the assessment of these factors for himself.

It is concluded that where the rheumatic child displays neurotic symptoms, as he or she infrequently does, these symptoms are superimposed on a nervous temperament and stand in no aetiological relation to the rheumatism. It is suggested that the personality of the rheumatic child shows a quantitative increase in emotion and kinesis—which has long been recognized by many clinical observers—and it is suggested that this nervous instability is an important factor in the development of the rheumatic state.

Treatment of the Nervous Rheumatic Child

There are two principles in the treatment of these children which are implicit in all that has been written here. The first is that the child, whether suffering from nervous disorder, growing pains, arthritis, or chorea, should not be regarded as suffering from the rheumatic state until the diagnosis of carditis is definitely established. The second is that evidence of nervous excitability or instability should be sought and, if found, treated in all such children before, during, and after the development of this rheumatic state. The first principle will prevent the formation of cardiac neurosis, and the second principle, I believe, gives the best hope of limiting the extent of the rheumatic disorder in childhood.

Nothing recondite is required in the way of psychiatric diagnosis and treatment for these children. They are nervous and timid, excitable and fearful, and the appropriate treatment is by way of reassurance, recognition, and solution, if possible, of environmental difficulty, and by sedatives.

Reassurance.—The need for reassurance should be remembered at every stage of treatment. Words spoken at the bedside should be definitely encouraging: no hint of diagnostic difficulty, of therapeutic failure, or of doubtful prognosis should be allowed to reach the child. Attempts should be

made by parents, doctors, and teachers to overcome the child's timidity and fear. He should be encouraged by every means to believe that his background is stable and his environment secure. The best means to this end is to surround him with a confident family circle. The already anxious mother will be quick to catch the physician's dominating fear, which is the fear of relapse, and the family atmosphere seldom brings reassurance to these children. Fortunately, most of these children are much less fearful of the effect of physical activity than are their mothers and doctors.

Winnicott (1938) has written: "It may be said that once we have diagnosed rheumatism implying that the child is liable to heart disease our job is to produce in the child an *induced hypochondria.* This is our first task, and the future depends on our success rather than on drugs." No doubt this holds a certain element of truth, but it also holds a greater element of danger. Hypochondria will not help to return the child to normal living, and this last must surely be the object of all therapy. It is important to recall constantly, when treating rheumatic heart disease in children, that a cardiac neurosis is frequently imposed on these children. Paul White (1937) reported that 4.7% of patients with organic heart disease had "neuro-circulatory asthenia"; and Paul Wood (1941) found that out of 100 recruits with early organic heart disease 13 showed the symptoms of Da Costa's syndrome. I believe that once the active rheumatic process has subsided (and of this we can to-day furnish good evidence with the sleeping pulse rate and the sedimentation rate) the child should be returned to normal living as quickly as possible, and its activity should then be limited, not by some imposed standard but only by the heart's capacity. I believe that nervous relapse (chorea and subacute rheumatism) is best prevented by the methods outlined here. The prevention of further pharyngeal infection presents a difficult problem. The removal of large numbers of these children to Florida has been shown not to prevent it. Schlesinger (1938) has shown that continuous aspirin therapy reduces the liability to streptococcal relapses: it is probable that continuous sulphonamide therapy will prove itself more effective (Thomas, 1942). I believe that no measures should be taken to prevent infection which interfere with the child's normal living.

Environmental Difficulties.—These may be related to the family or to school. In neither case is removal from the family or prolonged absence from school likely to offer a permanent solution. It is amazing how light-heartedly children are kept away from school. The child that cannot adapt itself to its family has failed at its first hurdle, the child that cannot adapt itself to the normal educational stresses has failed at its second, and such children are unlikely to succeed at the later stages of the race. An attempt should be made to iron out the grosser environmental stresses, but the aim should always be to alter the child's abnormal reactions to its environment.

The Value of Sedatives.—I believe that continuous sedation by phenobarbitone is first-class therapeutics for these children with growing pains and the subacute rheumatic state, with chorea, and with active rheumatism. It will no doubt be objected that there are dangers in this continuous treatment by sedatives. I can only say in reply that I have watched these children for several years and am convinced that their physical and mental performance is improved by such therapy.

Growing Pains.—The child is depressed, weeps easily, sleeps badly, and is worried by pains. In time its anxieties appear to be related entirely to its physical symptoms. "There, I'm flushing again: My heart's beating away: I can't eat my dinner: I've got that pain in my legs: Why can't I stop twitching?: I shall never get to sleep." These are the cries of the child, and phenobarbitone causes the symptoms to disappear.

Chorea.—I believe that every child who has had chorea should be treated with phenobarbitone daily until after puberty. I have had 12 unselected children who have suffered from chorea under continuous observation and such treatment for periods varying from three to six years. Only one of them has had a relapse of chorea and only two have had another rheumatic episode in the period of observation. This is much less than the expected relapse rate for such children. Other observers have found that it is possible to reduce the expected relapse rate in chorea by the attempt to remove environmental stress but without continuous sedation. Gerstley and his collaborators recorded a relapse in only 6 children out of 45 with chorea over a period of seven years, and "three of those had not been faithful attendants at the clinic." Their prophylactic treatment was "by keeping the youngsters under constant observation, obtaining co-operation from the school teacher, and avoiding the radio and thrilling movie."

The Rheumatic State.—All children with acute rheumatism should be given sedatives, since all are liable to chorea. Such therapy is more important for girls than for boys, and more important for children who have had previous attacks of chorea. As to the need for sedation in the other phases of the rheumatic state, the physician must assess the instability of the individual child. I have formulated for my own use the following rules for the administration of phenobarbitone to these children. It should be given to all rheumatic children who (1) show undue excitability, timidity, or motor instability; (2) show disorders of sleep, digestion, or appetite; (3) complain of fatigue or other "circulatory" symptoms unrelated to physical activity (i.e., the common complaint of tiredness in the morning). As an example of the results of this treatment the following case is related:

A girl aged 12 was admitted to the hospital for the first time in April, 1941. She had been known to have a "weak heart" since an examination by the school medical officer at the age of 5. She had not been allowed to play games, go to the gymnasium, swim, or cycle. She has always slept badly and had a poor appetite. Her weight was 4 st. 12 lb. before her present illness. Her mother said she was very excitable and, although usually happy, was liable to violent outbursts of temper. These outbursts were generally related to imposed restriction of her physical activities. She had an attack of rheumatism after measles at the age of 8, but had had no other rheumatic episode since. Examination of her heart showed pericarditis and fully developed mitral stenosis. Her sedimentation rate was normal in six weeks, and she was allowed out of bed in ten weeks. She returned home and has had 1/2 gr. of phenobarbitone night and morning since. She sleeps well, has a good appetite, and has gained 20 lb. in the last year. She is said to be happy, even-tempered, and much less excitable. She rides a bicycle, has learnt to swim, has spent a week under canvas at a girls' camp, and has had no time away from school.

It is suggested that the treatment of these children by simple psychotherapy and by sedation removes their growing pains, reduces relapse in chorea, and improves the mental and physical performance of the rheumatic child.

Summary of the Argument

The rheumatic state in childhood has two main components—nervous excitability and infection. These two factors are present in varying degree in any one rheumatic child, so that it may be impossible in one child to distinguish any nervous instability or in another to detect any certain evidence of infection. In one, the profound motor and emotional instability which is called chorea may possess the child and infection be either absent or a late invader; while in another, infection dominates the scene at the abrupt onset and throughout the subsequent course.

It is concluded that growing pains are usually the result of some difficulty attending the growth of personality, not of stature, and that it is more easy to find evidence of nervous instability than of infection in the subacute rheumatic state.

The nervous instability of the rheumatic child has no relation to neurosis; it is suggested that it is a quantitative increase in emotion and kinesis. The relationship between the nervous excitability and the infection is difficult to determine. It has been generally assumed that such nervous instability as is allowed to the rheumatic child is the direct result of the "rheumatic infection or toxaemia," and this view is supported by the fact that chorea may develop for the first time during an attack of acute rheumatism. There is more evidence to support the contrary view that this nervous excitability in the child commonly precedes the onset of infection and may even prepare the ground for the rheumatic invader.

The various aetiological factors in the rheumatic state are potent in so far as they predispose to these two components—nervous excitability and infection. Age, sex (as in chorea), and the nervous stresses of poverty are important since they contribute to the appropriate nervous excitability; climate, cold and damp, overcrowding, and the other physical accompaniments of poverty are important because they increase the liability to droplet infection. The heritable factor in the

rheumatism of childhood cannot be of great importance, since there are many facts that declare it primarily an environmental disorder.

Glover (1930) has called rheumatism an obsolescent disease, and this no doubt depends on the slow removal of the appropriate environmental stresses amongst the poor. Such "gradualness may be inevitable," but it is suggested that the health of the rheumatic child is improved and stabilized by treatment of its nervous instability. This is done by the removal of the grosser environmental stresses, if possible, by the adjustment of the child's reaction to its environment, and, most important of all, by continuous sedation with phenobarbitone.

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REFERENCES

Bradley, W. H. (1932). *Quart. J. Med.*, **25**, 79.
Buzzard, E. F., and Greenfield, J. G. (1921). *Pathology of the Nervous System*, London.
Cameron, H. C. (1929). *The Nervous Child*, London.
Cheadle, W. B. (1889). *The Rheumatic State in Childhood*, London.
Coombs, C. F. (1924). *Rheumatic Heart Disease*, Bristol.
Fernando, P. B. (1939). *Quart. J. Med.*, n.s., **8**, 261.
Findlay, L. (1931). *The Rheumatic Infection in Childhood*, London.
Freeman, W. (1933). *Neuropathology*, Philadelphia.
Gerstley, J. R., *et al.* (1935). *J. Pediat.*, **6**, 42.
Glover, J. A. (1930). *Lancet*, **1**, 499.
Gordon, R. G. (1939). *A Survey of Child Psychiatry*, p. 5, London.
Hawksley, J. C. (1939). *British Medical Journal*, **1**, 156.
Holt, L. E., and McIntosh, R. (1940). *Diseases of Infancy and Childhood*, New York.
Kaiser, A. D. (1927). *J. Amer. med. Ass.*, **89**, 2239.
—— (1934), Ibid., **103**, 886.
London County Council Annual Report, 1936, **3**, 44.
Medical Research Council (1927). Spec. Rep. Series No. 114.
Morris, J. N., and Titmuss, R. M. (1942). *Lancet*, **2**, 59.
Neustatter, W. L. (1937). *Guy's Hosp. Rep.*, **87**, 8.
Poynton, F. J. (1936). *Harben Lectures*, London.
Schlesinger, B. (1938). *Lancet*, **1**, 649.
Seham, M., and Hilbert, E. H. (1933). *Amer. J. Dis. Child.*, **46**, 826.
Shapiro, M. J. (1939). *J. Pediat.*, **14**, 315.
Sheldon, W. (1936). *Diseases of Infancy and Childhood*, London.
Sherrington, C. S. (1940). *Man on his Nature*, Cambridge.
Still, G. F. (1912). *Common Disorders and Diseases of Childhood*, London.
—— (1927). Med. Research Council Report, vide supra.
Swift, H. F. (1940). *J. Amer. med. Ass.*, **115**, 1509.
Thomas, C. B. (1942). *Bull. N.Y. Acad. Med.*, **18**, 508.
White, P. D. (1937). *Heart Disease*, New York.
Wilson, S. A. Kinnier (1928). *Modern Problems in Neurology*, London.
Winnicott, D. W. (1931). *Clinical Notes on Disorders of Childhood*, London.
—— (1938). In *A Survey of Child Psychiatry*, p. 28, Oxford.
Wood, P. (1941). *British Medical Journal*, **1**, 846.

MACROCYTIC ANAEMIA OF PREGNANCY AND THE PUERPERIUM

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It is generally believed that the macrocytic anaemia, or so-called "pernicious anaemia," of pregnancy is due to a lack of the anti-pernicious-anaemia principle brought about by one or more of the following causes: (*a*) a temporary depression of secretion of the specific intrinsic factor of Castle (Strauss and Castle, 1933; *Brit. Ency. med. Pract.*, 1936); (*b*) a deficient intake of the extrinsic factor (Strauss and Castle, 1933; *Brit. Ency. med. Pract.*, 1936); and (*c*) a diversion to the foetus of the anti-anaemic principle itself (Vaughan, 1936). The usual conception of treatment is that it is similar to that of Addisonian pernicious anaemia except that in the macrocytic anaemia of pregnancy it may be discontinued after delivery, when the demands of the foetus no longer exist and a return of the secretion of intrinsic factor has been presumed to occur (Strauss and Castle, 1933; Vaughan, 1936). The three cases to be described are of interest because there was little or no improvement in the blood level after liver extracts given parenterally, but rapid regeneration occurred when this treatment was supplemented by the ingestion of whole liver.

Case I

Mrs. A., aged 39, was admitted to hospital in Nov., 1941, during the 36th week of pregnancy. For two weeks before admission she felt tired and breathless and vomited frequently. The pregnancy had until then been uneventful, and she had taken a diet which was notable in only one respect—namely, that she had never cared for and seldom ate any red meat. She had five children, all alive and well. The last child was born in Nov., 1937, and at that time she was noted to be severely anaemic and was given a blood transfusion after labour. Next day the haemoglobin was 30% and a film was described as showing the features of pernicious anaemia. In the next 13 days 40 c.cm. of campolon was injected. Reticulocyte counts apparently were not done, but it is of interest that 15 days after the first injection the haemoglobin had fallen slightly to 28%. Large doses of campolon were continued, and recovery was then rapid. She was discharged with a haemoglobin of 70%; no further treatment was given. There seems little doubt, therefore, that she had had "pernicious anaemia" of pregnancy in 1937.

When seen in Nov., 1941, she was obviously severely anaemic. Physical examination revealed no notable abnormality. The tongue and nails were normal. Free hydrochloric acid was secreted after the injection of histamine—maximum, 30 units at one hour. The haemoglobin was 20%, and the other findings were compatible with Addisonian pernicious anaemia except that anisocytosis was less than would be expected at this level. A Price-Jones curve gave a mean cell diameter of 8.5 μ.

In the first three days 10 c.cm. of campolon and 8 c.cm. of anahaemin were injected (see Fig. 1). Three days after the first injection there was a reticulocytosis of 9.7%. On this day delivery occurred and a blood transfusion of two pints was given. The blood loss after delivery was estimated at about one pint. The slight improvement produced by the transfusion was transient and the blood count had fallen almost to the initial level on the tenth day. From the eighth day onwards daily injections of anahaemin or neo-hepatex and small doses of campoferron orally were given. On the sixteenth day there was a reticulocyte crisis of 40%, but very little rise in the red cell count occurred. On the fourteenth day treatment with whole liver was begun. The reticulocytes were 29% on the thirtieth day, and the blood count then rose rapidly.

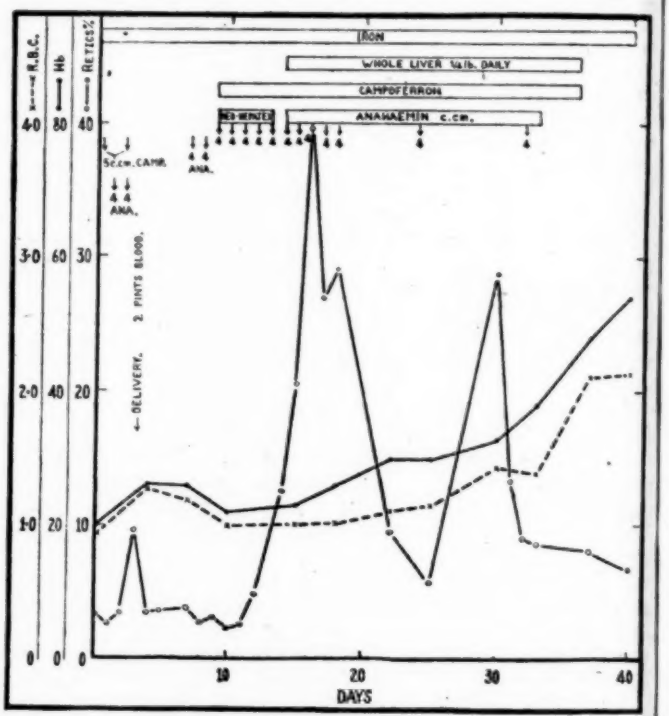

FIG. 1.—Chart of Case I.

Case II

Mrs. B., aged 39, was admitted to hospital in June, 1941, five weeks after giving birth to her sixth child. She insisted that she had no untoward symptoms until three weeks after delivery, when she began to be breathless and tired; but she was inclined to belittle her symptoms, and it is quite possible that their onset was earlier than this. At the same time there developed a burning pain in the tongue, and her appetite, which previously had been excellent, began to fail, so that shortly before admission she had literally to force herself to eat. Her diet appeared to have been satisfactory, and was

distinctly better than that taken by most women of the hospital class.

The tongue showed no papillary atrophy, but there was a superficial ulcer on each margin. The spleen was easily palpable four fingerbreadths below the costal margin. Free hydrochloric acid was secreted after the injection of histamine. The initial blood count was: Hb 27%, R.B.C. 1,400,000, C.I. 0.96, reticulocytes 3.7%, W.B.C. 3,100 (see Fig. 2). The appearances in a film were compatible with Addisonian pernicious

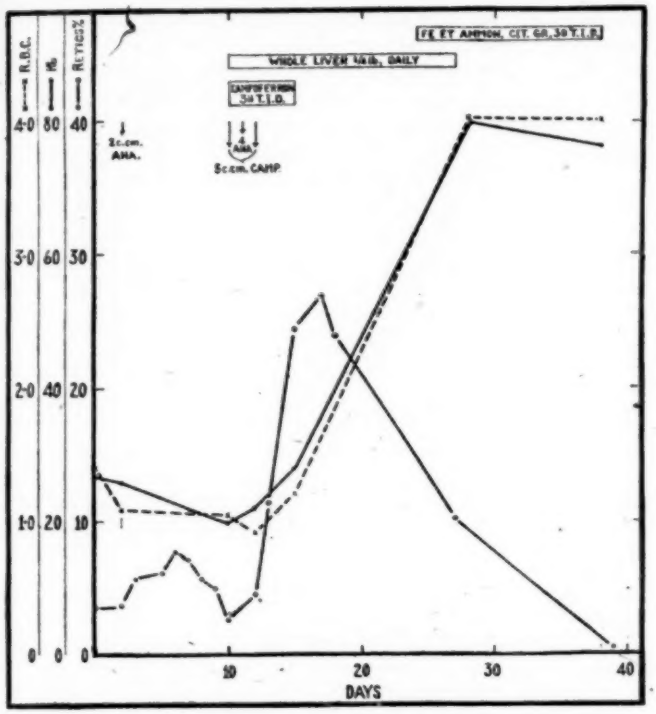

FIG. 2.—Chart of Case II.

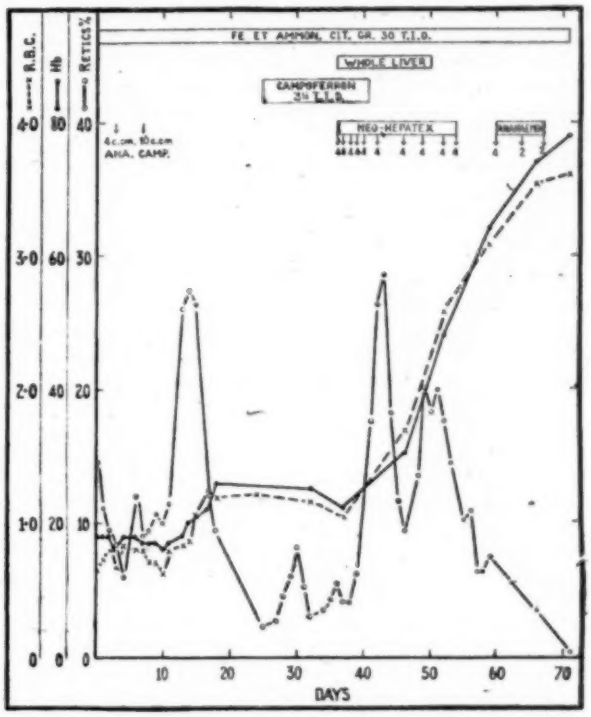

FIG. 3.—Chart of Case III.

anaemia. Sternal marrow showed 55% of erythroblasts, with 18% of megaloblasts. Following injection of 2 c.cm. of anahaemin there was a small reticulocyte response (maximum 7.6% on the fourth day), but in spite of this the blood count fell slightly. On the seventh day after beginning treatment with whole liver, campoferron, campolon, and anahaemin the reticulocytes reached a peak of 27%, and this was followed by extremely rapid blood regeneration, the haemoglobin rising by 60% and the red cells by 3 millions in the 18 days following the start of this treatment. The patient has been seen at intervals for six months, and has maintained a normal blood count without further treatment.

Case III

Mrs. C., aged 36, was admitted to hospital in Sept., 1941, 6 weeks after giving birth to her ninth child. Immediately after labour she felt weak and tired, and these symptoms gradually became more marked. For about a week before admission there were diarrhoea and pain in the tongue. Her appetite was good until the last two months of pregnancy, and she had always taken a good mixed diet. In this case there was histamine-fast achlorhydria, and the tongue papillae were atrophic. The initial blood count was: Hb 18%, R.B.C. 650,000, C.I. 1.30, W.B.C. 3,600, reticulocytes 14% (see Fig. 3). The film was indistinguishable from that of Addisonian pernicious anaemia. A Price-Jones curve gave a mean cell diameter of 7.88 μ, and the mean cell volume was 92.6 cu.μ. The sternal marrow showed 28% erythroblasts, including 8% megaloblasts.

A reticulocyte response of 27.5% occurred 11 days after the injection of 4 c.cm. of anahaemin and 7 days after 10 c.cm. of campolon. The blood count, however, rose only slightly, and then gradually fell. Seven days after beginning treatment with whole liver and daily injections of neo-hepatex there was a reticulocytosis of 28.6%, and another peak of 20% occurred 6 days later. At the same time the blood count began to rise very rapidly.

Discussion

That the macrocytic anaemia of pregnancy and the puerperium may be refractory to treatment which is efficacious in Addisonian anaemia is not well recognized, and it is not clear whether this fact is due to its rarity or whether it has been largely overlooked. A study of the literature shows that cases of this type have occurred (Stevenson, 1937-8; Ungley, 1938); but little emphasis has been laid upon them, and in a recent review by Sodeman (1940) they receive no mention. In the discussion which followed a communication based on the cases reported here (meeting of the Association of Physicians of Great Britain and Ireland, April, 1942), L. S. P. Davidson described a series of 16 cases of macrocytic anaemia occurring in pregnancy and the puerperium of which 10 were temporarily refractory to treatment with massive doses of liver extract. Details of these cases have been published by Davidson, Davis, and Innes (1942). Now that attention has been drawn to cases of this type it is possible that they will be found to form a considerable proportion of the macrocytic anaemias associated with pregnancy.

It is unfortunate that in each of the cases reported above the degree of anaemia was so severe that the separate trial of different therapeutic agents was not justified. Accordingly, massive treatment with parenteral liver extracts, campoferron (oral liver extract with iron, Bayer Products Ltd.), and whole liver was instituted in each case after the effect of parenteral liver extract had been found to be slight or absent. This makes interpretation of the results more difficult than would have been the case if the different forms of therapy had been used separately, but analysis of the results appears to justify certain conclusions.

In Cases I and III large doses of campolon (Bayer Products Ltd.) and anahaemin (British Drug Houses Ltd.) produced reticulocyte responses but little or no rise in the blood count, while in Case II 2 c.cm. of anahaemin brought about a small reticulocyte response but no improvement in the anaemia. All the liver extracts used were potent; for example, the same dose of the same batch of anahaemin as was used in Case II produced an optimal reticulocyte response and rapid blood regeneration in a case of Addisonian pernicious anaemia. Moreover, there was no evidence of sepsis or other condition that might have had an inhibitory effect, and the rapid blood regeneration which followed massive therapy makes it clear that marrow hypoplasia was not present. It may be concluded, therefore, that a deficiency of factors other than the anti-pernicious-anaemia principle was concerned in the production of the anaemia. The evidence suggests that the ingestion of whole liver provided these factors. It might be argued that they were present in low concentration in the parenteral extracts

used, and that these extracts in the very large doses given were responsible for the ultimately satisfactory blood regeneration. This possibility seems unlikely, since Cases I and III showed little or no rise in the blood count after quite large initial doses of campolon and anahaemin. Moreover, the concentration of such additional factors in liver extract is likely to be much greater in crude preparations like campolon than in the refined extracts anahaemin and neo-hepatex (Evans Sons Lescher and Webb), and it is seen from Figs. 1 and 3 that campolon was not given during the period of massive therapy which produced rapid blood regeneration. The effect of campoferron can probably be discounted, since the dose given was small, and when used alone for 11 days in Case III this preparation produced a small reticulocytosis but no rise in the blood count.

Attention is drawn to the double reticulocyte response which occurred during the period of massive therapy in Cases I and III. The phenomenon was not discernible in Case II, perhaps because of the infrequency of the observations during this period. The time relationships of these reticulocyte crises suggest that the first was due to parenteral therapy, and the second to the ingestion of whole liver. The occurrence of this second reticulocytosis, and the fact that it was accompanied and followed by rapid blood regeneration, are further evidence that whole liver provided essential factors which were not present in the parenteral extracts.

The characteristics of the responses shown by the cases under discussion suggest an analogy with the macrocytic anaemia of sprue and other steatorrhoeas, in which it has been noted that in some cases blood regeneration does not follow the reticulocytosis produced by parenteral liver extracts unless a high protein diet is also given. Davidson (1939) has described a case of this type and suggested that the high intake of protein might be necessary for the formation of red cell stroma. Of particular interest is the fact that one of the constituents of the diet used in his case was 1/2 lb. of liver daily. Moreover, I have recently studied 3 cases of macrocytic anaemia associated with steatorrhoea which were refractory to parenteral extracts but responded rapidly when whole liver was given without any other alteration in the diet. It is hoped to publish these cases at an early date. These observations suggest that in cases of macrocytic anaemia of pregnancy and the puerperium of the type here described there may exist a deficiency of factors present in whole liver similar to those which are lacking in some cases of macrocytic anaemia associated with steatorrhoea. The nature of these factors and the causes which produced deficiency in the cases described are quite obscure. Certainly the histories do not point to direct dietary deficiency, and although a temporary depression of absorption from the intestine brought about by the pregnancy would explain the findings, there is no direct evidence that such a condition existed.

Summary

Three cases of macrocytic anaemia of pregnancy and the puerperium are described. The results of treatment showed that deficiency of the anti-pernicious-anaemia principle was not the only factor concerned in the production of the anaemia. Reticulocytosis after injection of liver extract may not be followed by remission in the macrocytic anaemia of pregnancy. The administration of whole liver appeared to provide other factors necessary for blood regeneration. An analogy is drawn between these cases and the macrocytic anaemia associated with steatorrhoea.

REFERENCES

British Encyclopaedia of Medical Practice, 1936. **1**, 441, Butterworth.
Davidson, L. S. P. (1939). *Edinb. med. J.*, **46**, 474.
—— Davis, L. J., and Innes, J. (1942). *British Medical Journal*, **2**, 31.
Sodeman, W. A. (1940). *Amer. J. med. Sci.*, **200**, 117.
Stevenson, E. M. K. (1937–8). *Trans. Edinb. obstet. Soc.*, **58**, 81.
Strauss, M. B., and Castle, W. B. (1933). *Amer. J. med. Sci.*, **185**, 539.
Ungley, C. C. (1938). *Lancet*, **1**, 925.
Vaughan, J. M. (1936). *The Anaemias*, p. 121. Oxford Med. Publications, London.

Taking advantage of the investigation by bronchoscopy of patients suffering from various pulmonary lesions, L. Tuft and N. M. Levin (*Amer. J. med. Sci.*, 1942, **203**, 717) took samples of bronchial secretion before and after the intravenous injection of sodium iodide. The iodide was detected in the bronchial secretion within 15 to 20 minutes after intravenous injection. The authors believe that the excretion of iodides into the bronchial tree accounts for their expectorant action.

ECONOMY IN FATS AND OILS IN TREATMENT OF SKIN DISEASES

BY

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In September, 1939, it was realized that the shortage of fats and oils which would inevitably occur might in time seriously affect the treatment of diseases of the skin. It was then recommended that wet dressings and varnishes should be used whenever possible in place of ointment and paste preparations. Such substitution is not, however, universally applicable. Just as some types of inflammatory skin diseases, particularly inflammatory processes of the eczematous type, are intolerant to greasy applications of all descriptions in certain phases of their course, so other inflammatory conditions, including most cases presenting the eczema type of inflammation, demand grease in some form if a response to treatment is to be achieved. These well-known facts can only be interpreted as showing that processes or reactions which occur at the point of contact between the epidermal surface on the one hand and the opposing surface of the vehicle containing the therapeutic agent on the other have a profound influence on the course of the disease, and this quite apart from any known pharmacological action of substances incorporated in the vehicle. Greasy bases are therefore essential and irreplaceable in dermatological therapy.

A substantial quantity of a greasy base must be used in the treatment of a case. For example, 2 lb. of a paste containing 15% zinc oxide in paraff. moll. is necessary for a single adequate dressing of an eczematous eruption involving the entire body, 6 oz. for an arm and hand only, and 3 oz. for a hand and fingers only. Less is required if zinc oxide or other inert powder employed to give the paste consistence is omitted; but it has been pointed out that the nature of the base may be all-important and determine success or failure with an incorporated drug, and it frequently happens that the paste consistence is essential. To remove a paste from the inflamed skin a preliminary softening and cleansing with a mineral or vegetable oil is necessary, followed by washing with soap and water. The oil and soap add to the total amount of fats and oils used in the treatment of the disease.

As there is no substitute for grease, economy in its use can only be achieved by some procedure or manipulation that eliminates the necessity for using oil and soap in conjunction with it, and this can be done.

Skin-cleansing Substances

The following substances can be added to a bath in small amounts or used in the same way as liquid soap; they will remove from the skin a paste containing 15% zinc oxide without a preliminary cleansing of the skin with oil:

Sodium salt of highly sulphonated oleic acid (M 1337)*
Polyethenoxy-ether of cetyl alcohol (M 1344)
The sodium salt of the sulphuric ester of sperm oil alcohol (M 1773)
Cetyltrimethyl ammonium bromide

These substances have been used as cleansing agents with no deleterious effect on acutely eczematous skin. They are not primary irritants and, except in the rarest instances, are not substances to which idiosyncrasy may be expected. The latter statement can of course be applied to any widely used skin application or skin contact substance, either therapeutic or cosmetic, or to wearing apparel.

The removal of a pomade with a soft paraffin base which has been thoroughly rubbed into the hair and scalp in the female is an extremely arduous and frequently disappointing procedure when soap preparations are used, but these substances can completely and easily cleanse such a scalp.

* These substances are standardized mixtures to which it would be difficult to assign a precise chemical definition, and for this reason a number is appended to a general descriptive nomenclature.

Emulsifying Agents

Another method of economizing in oil is to incorporate an emulsifying agent in the ointment base. The following formulae have been tried:

	Parts
℞ Paraff. moll.	25
Octadecyl alcohol	2–4
Diethylene glycol distearate	2.5–5
Sodium cetyl sulphate (15% paste)	1
Water	22–26

This emulsion must be kept in a closed container to avoid evaporation.

℞ Tetraethenoxy-cetyl alcohol	10–20 g.
Paraff. moll.	ad 100 g.

℞ Cetyl alcohol — 4 mols ethylene oxide ..	40	parts, of this 10–20 g.
Cetyl alcohol	40	
Sodium cetyl sulphate ..	20	
Paraff. moll.	..	ad 100 g.

To these preparations may be added any of the ingredients commonly incorporated in the ointment bases—i.e., zinc oxide, ichthammol, mercury, sulphur, etc.

These three formulae have been used with the addition of 15% zinc oxide and an active ingredient such as ichthammol or ammoniated mercury in the treatment of a sufficient number of eczematous skin eruptions to give convincing evidence that they fulfil the same conditions as regards suitability as was found in the case of the cleansing agents. To remove these preparations from the skin no oil is required, and considerably less soap is used than is necessary when a cleansing oil has been applied before the soap and water in the removal of an ordinary paste base; sometimes it is possible to dispense with soap altogether.

Comment

In the case of the cleansing agents, oil and soap are dispensed with; in the case of the emulsifying bases, oil is not necessary and little or no soap is used. The extent of the economy which might result from the employment of these preparations or others of similar nature can be judged when the oil and soap consumption of a large department for the treatment of skin diseases is examined. The weekly requirement in one such department containing 60 beds and catering for the daily treatment of from 20 to 30 out-patients referred from the consultation-room is about 400 oz. of oil and 12 lb. of soap. In addition to economy in actual material there is a considerable reduction in the time required to carry out treatment. To prepare for a bath a case in which a paste has been applied to the entire body surface at least half an hour's work with oil is necessary, and at least part of this operation requires the services of a nurse.

Finally, the removal of ordinary pastes necessitates a considerable amount of friction, which has some temporary deleterious effect on the course of an eczematous eruption. The employment of either type of preparation described above obviates this drawback.

My thanks are due to Imperial Chemical Industries (Pharmaceuticals) Ltd., Manchester, for the preparations used in this investigation.

THE BREAD FACTOR IN A WARTIME HUMAN DIETARY

BY

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Contradictory statements have appeared in the literature as to the merits or demerits of national wheatmeal bread and of the proposals to fortify this bread with calcium in the form of creta praeparata or white bread with aneurin (vitamin B_1) and calcium in the same form. It was thought, therefore, to be of interest to record the results of some feeding experiments recently carried out with rats.

The experiments were designed to determine the effect on the growth of young rats of substituting national wheatmeal bread or fortified white bread for the ordinary white bread in the basal diet. This basal diet was drawn up from the results of a dietary survey carried out in a North of Scotland town in the spring of 1941, and represents the average diet of a number of families spending up to 7s. per head weekly on food. The rats' basal diet contained all the various foods in the proportion in which they were found in the human diet. The methods used in the preparation of the food, which was in all cases fed *ad lib.*, were similar to those described for an earlier dietary experiment at this institute (Orr, Thomson, and Garry, 1936). Since "fortified" white bread was not available in this part of the country, white bread supplemented with chalk and aneurin in the proportions officially proposed was employed for this purpose.

Experiment I

In the first experiment six groups of rats were used, and the diets and weight increases for each group over a period of eight weeks are shown in the accompanying table. The

Table showing Diets and Weight Increases for Experiments I and II

Group	Diet		Males			Females		
	Type of Bread	Supplement	Initial Weight	Weight After 8 Weeks	Average Weekly Gain	Initial Weight	Weight After 8 Weeks	Average Weekly Gain
			g.	g.	g.	g.	g.	g.
			Experiment I					
1	White	—	42·4	191·9	18·7	40·4	149·4	13·6
2	N.W.M.	—	44·6	208·4	20·5	40·2	151·1	13·9
3	White	Ca+aneurin	43·7	228·2	23·1	42·1	168·0	15·7
4	N.W.M.	Ca	42·6	231·3	23·6	41·1	165·5	15·6
5	White	Vitamins+minerals	43·7	227·3	23·0	40·8	166·9	15·8
6	White	Ca	40·3	205·9	20·7	38·5	156·7	14·8
			Experiment II					
2	N.W.M.	—	40·5	208·2	21·0	39·5	157·9	14·8
3	N.W.M.	Ca+aneurin	40·3	242·7	25·3	39·5	186·4	18·4
4	N.W.M.	Ca	40·3	246·9	25·8	39·9	183·6	18·0
5	N.W.M.	Vitamins+minerals	40·3	259·1	27·4	39·2	192·5	19·2

rats were given their respective diets at weaning, approximately half of them being taken from the stock colony, the others being the offspring of parents which had been reared on the corresponding experimental diets in a preliminary experiment. Each group contained 14 or 15 males and 15 females, the sexes being caged separately. The mineral–vitamin supplement used in Group 5 contained calcium, phosphorus, iron, and vitamins A, B_1, and C in amounts such as would, if fed in proportion to the quantity supplied to the rats, have brought the daily intake of the members of the families surveyed up to Ca, 1 g.; P, 1.3 g.; Fe, 15 mg.; vitamin A, 4,000 I.U.; vitamin C, 1,500 I.U.; and vitamin B_1 390 I.U. (i.e., ratio to calories 1:5).

The figures show that for the male rats the average weekly gain of 18.7 g. in the control group has been increased to 20.5 g. by the substitution of national wheatmeal for white bread. A similar increase (20.7 g.) was found in the group which received white bread with a calcium supplement. A greater improvement was found in the groups which received both additional calcium and vitamin B_1, whether the B_1 was given as aneurin or as national wheatmeal bread. In these groups the average weekly gains were 23.1, 23.6, and 23.0 g. Similar results hold also for the groups of females, except that the substitution of wheatmeal bread for white had little effect on the rate of weight increase. The marked improvement caused by simultaneous addition of calcium and vitamin B_1 was again observed.

Experiment II

Experiment II was intended to be a second-generation repetition of Experiment I, all the young rats in this test being the offspring of animals used in the growth tests just recorded. Unfortunately white bread was no longer available, so some modification of the groups was necessary. It was decided to test the effect of increasing the ratio of I.U. of vitamin B_1 to calories by adding extra aneurin. In Groups 2 and 4, which correspond to the groups of the same numbers in Experiment I, this ratio was 1:4.33. The amount of aneurin used as supplement for Group 3 brought this ratio up to 1:3, and the ratio for Group 5, with a mineral+vitamin supplement as in Experiment I (but with the complete B complex instead of B_1 alone), was 1:4. In this experiment each group contained 17 or 18 males and a like number of females.

The figures for the weekly gain in weight again show the beneficial effect of the calcium supplement. Thus for the males the average gain of 21.0 g. on the unsupplemented ration was increased to 25.8 g. by the addition of calcium, while for the females the figures were 14.8 and 18.0 respectively. The increased amount of aneurin given to Group 3 had little or no effect on the growth of the rats. The best response was shown by the animals in Group 5, which received the complete vitamin-mineral supplement. Here the gains averaged 27.4 and 19.2 g. for males and females respectively; but the greater part of the extra increase was undoubtedly due to the added calcium, as is shown by comparison with the results for Group 4.

Comment

These results point quite definitely to the fact that the addition of calcium to this diet in the amount which it has been proposed to add to national wheatmeal flour does not produce any harmful results, but, on the contrary, tends definitely to enhance the value of the diet as measured by the live-weight increase of young rats. On these figures there is no evidence in support of further additions of vitamin B_1 in the form of aneurin when national wheatmeal bread is used. In so far as they go, the data just cited support the findings of Gaunt, Irving, and Thomson (1938) that a poor human dietary of the type used in this experiment can be materially improved by supplementing with calcium in inorganic form.

REFERENCES

Gaunt, W. E., Irving, J. T., and Thomson, W. (1938). *British Medical Journal*, 1, 770.

Orr, J. B., Thomson, W., and Garry, R. C. (1935). *J. Hyg.*, Camb., **35**, 476.

Medical Memoranda

Anaesthesia for Laryngofissure

In view of the growing popularity of surgical treatment of carcinoma of the larynx it is important that an adequate anaesthetic technique should be evolved. In the past preliminary tracheotomy, with its attendant risk of chest complications, has usually been performed. The suggested method does away with the necessity for a tracheotomy and ensures post-operative haemostasis. In a recent series of 8 cases performed by Mr. Charles Keogh there were no post-operative chest complications.

Method.—Premedication: omnopon 1/3 gr. and scopolamine 1/150 gr. 1 hour before operation. Induction: anaesthesia is induced in the normal way with N_2O, O_2, and trilene, using a Boyle apparatus. When the patient is well into the third stage of anaesthesia a stiff gum-elastic intratracheal catheter, St. Bart's pattern, size 12–14, is passed through the larynx into the trachea under direct vision. Great care is taken to avoid the growth during this manœuvre. The proximal end of the catheter is now connected to the machine and anaesthesia is maintained with O_2 and trilene. Owing to the small bore of the tube it is usually necessary to supplement the volume of oxygen reaching the lungs by gentle intermittent pressure on the rebreathing bag with the expiratory valve fairly tightly screwed down.

Operation.—As soon as the incision has been made and the larynx opened the space between the catheter and the trachea is thoroughly packed off with narrow gauze, leaving one end emerging from the lower part of the wound. This ensures that no blood leaks down the trachea during the operation. The catheter is now found to lie in the posterior commissure of the larynx and usually interferes very little with the surgical procedure. If it proves to be in the way, then it may be retracted one way or the other. In only one case, where the growth was quite extensive, has this proved necessary. Anaesthesia is maintained as before with O_2 and trilene, aided by manual pressure on the rebreathing bag at each inspiration. Some of the gas percolates back through the packing in the trachea. As trilene is non-inflammable the diathermy may be used if necessary.

Haemostasis.—Following excision of the growth it is often very difficult to achieve perfect haemostasis. After removal of the tracheal packing and catheter the anaesthetist may play his part by passing a large-bore Magill tube, size 8–9, with inflatable cuff, through the nose and into the trachea under direct vision. When the cuff lies in the larynx it is inflated through the pilot tube until it occupies the lumen of the larynx and presses up against the field of operation. The laryngofissure is now sewn up, and the patient returns to the ward with a clear airway and a guarantee of haemostasis.

Post-operative Treatment.—The intratracheal tube is left in position for 8 hours. During this time the patient is given morphine, and a little 2% decicaine solution is sprayed down the tube from time to time. In only one case did the tube have to be withdrawn earlier than was desirable. In this case it had been passed through the mouth. Provided it is passed through the nose very little discomfort is experienced. Eight hours after the operation the laryngeal cuff is deflated. After waiting a few minutes the patient is asked to cough. If blood is coughed up through the tube, then the cuff is re-inflated and the same procedure repeated in 2 hours. If no blood appears, then the tube is gently but firmly withdrawn. Even this procedure causes remarkably little discomfort to the patient.

Conclusion.—The object of this technique is to provide safe anaesthesia for cases of laryngofissure without a tracheotomy and its accompanying disadvantages. Also it hinders the surgeon very little and assures him of post-operative haemostasis.

I am very grateful to Mr. Charles Keogh for his co-operation and valuable suggestions.

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A Combined Infection of the Throat with Diphtheria and Tuberculosis

I have failed to find in the literature any reference to such a combination, and am therefore of the opinion that the following case is worth recording.

The patient, a primipara about seven months pregnant, was admitted to Clayponds Isolation Hospital on Jan. 27, 1942, as suffering from diphtheria. The history given was that she developed a cold and cough on Dec. 27 and took to bed on Jan. 1. On Jan. 14 she had a sore throat, which she said was very swollen. On Jan. 26 vomiting began. Her doctor suspected diphtheria, and a throat swab taken that day was found positive for the diphtheria bacillus. On Jan. 27 she was given 16,000 units of diphtheria antitoxin intramuscularly before admission to hospital and a further 40,000 units intramuscularly after admission.

When admitted a greyish membrane was seen to cover the left side of the pharynx, left tonsil and pillars of the fauces, and the adjacent portion of the soft palate and uvula. The condition bore a resemblance to the smooth greyish-white appearance sometimes seen after a diphtheria membrane has separated, but did not appear to me to be quite typical. The left cervical glands were moderately enlarged and tender. The general condition of the patient was poor. Her face had a greyish cyanotic hue and the pulse was weak and rapid. The throat affection continued to spread, and it became evident in the course of a few days that the condition was one of extensive shallow ulceration with a thin ragged edge and a granulating base. The patient also had a distressing cough and brought up a few ounces of purulent sputum daily. Tuberculosis was suspected, and examination of the sputum on Feb. 3 showed that tubercle bacilli were present. Throat swabs taken on Jan. 30 and Feb. 8 still showed the presence of diphtheria bacilli. The Wassermann and Kahn reactions were negative. Physical signs in the chest were slight; but there was some impaired resonance, and air entry in the right scapular area. From Feb. 3 onwards the temperature was irregular, there being an average evening pyrexia of 100° F. The patient's appetite was poor, vomiting was frequent, and her general condition had deteriorated. On Feb. 14 she passed a few ounces of dark blood per rectum and also vomited some digested blood, becoming very collapsed. Though she revived somewhat, her condition on the following day was very poor, and on the morning of the 16th she began to complain of lower abdominal pain. At 2.30 p.m. the membranes ruptured, and at 3.30 p.m. she was delivered of a healthy premature male infant weighing 4 lb. 10 oz. Labour appeared comparatively easy, and was followed shortly by delivery of the placenta and membranes complete. There was no undue haemorrhage. At 5.15 p.m. the patient suddenly complained of inability to breathe. She became very collapsed and dyspnoeic, and died in about 10 minutes.

A post-mortem examination showed the presence of a thin-walled tuberculous cavity about 1¼ in. in diameter at the right apex. There was acute tuberculous bronchopneumonia, with numerous small ragged cavities occupying the remainder of the right upper lobe. The tuberculous ulceration of the throat extended over the epiglottis, giving it an eroded appearance, and oedema and granulation of the vocal cords were present. The heart was dilated and the muscle pale and flabby; the liver was enlarged and pale from cloudy swelling. No other abnormality was discovered. The uterus was healthy and well contracted.

The condition of the baby was satisfactory. It gained weight rapidly, and was discharged from hospital in a thriving state. The Mantoux reaction was negative up to 1 in 100 dilution.

The extent and the rapid spread of tuberculous ulceration in the upper respiratory tract seen in this case were quite unusual, and it is interesting to speculate whether a diphtheritic infection acted in this instance as an aggravating or a predisposing cause. The natural resistance to tuberculosis was obviously so poor that recovery was not to be expected. It was not anticipated, however, that the patient would die so suddenly. The strain of labour on a heart affected with diphtheritic carditis would help to explain the sudden death. As I had not had the opportunity of seeing this case in the earlier stages of the throat infection I cannot altogether exclude the possibility of the diphtheria bacillus existing merely as a saprophyte in a tuberculous throat, but nevertheless consider that the case is of unusual interest.

My thanks are due to Dr. Thomas Orr, medical officer of health for Ealing, for his kind permission to publish this case.

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Reviews

NASAL SINUSES

Nasal Sinuses. An Anatomic and Clinical Consideration. By O. E. Van Alyea, M.D. A William Wood Book. (Pp. 262; illustrated. 36s.) Baltimore: The Williams and Wilkins Company; London: Baillière, Tindall and Cox. 1942.

Dr. Van Alyea has written a book on the accessory sinuses, of which he has made a special anatomical study. He has combined with this clinical descriptions which consist largely of quotations from the published articles of numerous writers, showing evidence of wide if rather undiscriminating reading. The anatomical descriptions are good, but they reveal nothing in any way new or original, and the same applies to the excellent illustrations. It is to the anatomy of ethmoid bone that Dr. Van Alyea has chiefly devoted himself, and he has investigated the variations in its cellular structure in much detail. This is the most valuable part of the book and is its principal justification. The author says himself that no effort has been made in the limited space to cover all phases of sinus disease, but this hardly excuses the unequal treatment of the subject. Thus much space is devoted to lavage of the maxillary antrum, but the vaguest account only is given of the major operations on the accessory sinuses. The same applies to the description of osteomyelitis of the frontal bone: the reader is left with only a misty account of its pathology and how it should be treated. There is, nevertheless, a vast amount of information in the book, but it is scattered and incomplete.

The real purpose of the book is not clear, but it will be read with interest by specialists. The author, however, might have made it more interesting and informative if he had relied more upon himself, and refrained from the uncritical quotations of at least half of the authors to whom he has made himself indebted. In several places this degenerates into nothing more than a review of the literature, much of which is of an ephemeral character.

A FIFTY-YEAR-OLD TEXTBOOK

The Principles and Practice of Medicine. By Henry A. Christian, M.D., F.R.C.P. Originally written by Sir William Osler, Bart., M.D., F.R.C.P., F.R.S. Designed for the use of Practitioners and Students of Medicine. 14th (semicentennial: 1892-1942) edition. (Pp. 1,475. Cloth, 45s.) New York and London: D. Appleton-Century Company.

Fifty years ago the medical world was startled by the appearance of a textbook planned on really new lines. Although Osler was already physician-in-chief at Johns Hopkins he was little known in this country, except for his demonstration of platelets in the circulating blood. The book had an immediate success and conferred international fame on the author, whose personality was subsequently realized to be even greater than his book. The story of its origin and accomplishment is well told in Cushing's *Life of Sir William Osler*. It proved to be the last great medical textbook by a single writer, and in the subsequent editions produced by McCrae and later by Christian, two of his pupils, the Oslerian tradition has been so far as possible maintained and a continuity of thought secured.

One significant rearrangement has, however, been made. The book opens with a short introduction on psychosomatic medicine, which is justified on the ground that "in later years, with the enormous development of laboratory procedures and the increased utilization of complex techniques of examining the patient, the physician has found it increasingly easy to neglect the patient's personality and surroundings in his management of him, with the resultant feeling on the part of the patient that he is missing something that is traditional of the general practitioner who served his forebears." Psychosomatic medicine, often looked upon as new, is in reality "what the former generations of medical men practised without much use of names for it." Conformably with this outlook, the first section of this book is now devoted to functional diseases of the nervous system, to emphasize the fact that "every patient with organic disease presents a functional, emotional reaction which varies with the personality, the economic and social relationships, and the race and heredity of the individual."

It is a herculean task for one man to revise an account of the whole field of organic disease and bring it up to date. The editor rightly claims continuity of thought, but his claim to balance in description is perhaps not always so fully substantiated. Thus considerable space is given to rare and tropical parasitic diseases, while affections of the spleen are cursorily dealt with. However, so long as the encyclopaedic method prevails, the desire to include some reference to everything inevitably renders such balance precarious. For the rest we have nothing but praise, and we anticipate as much popularity for the book in its new form as it rightly had in the old.

HUMAN CONTROL OF CLIMATE

Climate and the Energy of Nations. By S. F. Markham, M.A., B.Litt., F.R.Met.S. (Pp. 144. 10s. 6d.) London: Oxford University Press. 1942.

The argument of Mr. Markham's pleasantly written and interesting little book is this. Modern research has demonstrated that the optimum working conditions indoors are given by an air temperature having a range of a few degrees above and below 70° F., with humidities ranging from 40 to 70%. Hence, in a social organization where artificial heating or cooling is unknown, optimum conditions will only be found near the annual isotherm of 70° F. But when men of genius, in a civilization able to profit from their genius, discover efficient means of warming and, eventually, cooling the atmosphere, so that maximal energy can be developed in colder (or warmer) natural climates, then the foci of civilization will or may move.

Mr. Markham sketches the history of civilizations and finds no facts inconsistent with this hypothesis. Human control of climate is a necessary condition of civilization. Perhaps Mr. Markham comes a little too near to suggesting that it is a sufficient condition, and has not allowed enough weight to the argument that disease, especially malaria, played a great part in sapping ancient civilizations. His rebuttal on p. 13 is *ad captandum*. Epidemiologically speaking, neither tuberculosis nor cancer, however energy-destroying, can have the nation-wide or continent-wide effects of malaria or hookworm disease. The book is well worth reading.

Notes on Books

The very practical textbook *A Glasgow Manual of Obstetrics*, by S. J. CAMERON, JOHN HEWITT, and ELLEN D. HEWITT, is now in its fourth edition, the last three having been published in the short space of 6 years. Its popularity is well merited. The new edition, which is larger than its predecessors, records most fresh developments in the field of obstetrics while emphasizing the need for sound conservative methods. Two surprising omissions are that in the treatment of eclampsia credit is not given to the Dublin school for seeing the light and abandoning their 40-year practice of colonic lavage, and no reference is made to the condition of erythroblastosis, with which the modern student should be familiar. This very sound book is published by Edward Arnold and Co. at 25s.

The fifteen years that have elapsed since the first edition of Mr. H. M. TRAQUAIR'S *Introduction to Clinical Perimetry* appeared have seen considerable additions to our knowledge both of the normal field of vision and of pathological changes in it. Perhaps the most noteworthy developments have been angioscotometry and the clearer recognition of opto-chiasmic arachnoiditis. Colour fields at one time attracted considerable and perhaps undue attention, and it is a measure of Traquair's knowledge, balanced judgment, and powers of exposition that the plan of his book has hardly changed throughout the four editions it has now reached. The present edition carries amplifications and additions derived from the more recent literature, and the book remains the indispensable guide for ophthalmologists and neurologists that it has been from the beginning. It is published by Henry Kimpton at 30s.

Of several books on sulphonamide chemotherapy WESLEY W. SPINK'S *Sulfanilamide and Related Compounds in General Practice* is, we believe, the first to appear in a second edition. It has been rearranged and considerably enlarged, the chief subjects more fully dealt with being the general principles of treatment and the mode of action of these drugs, their local application, and the treatment of intestinal infections. The book contains much information of great practical value, and is as nearly up to date as a publication on a rapidly progressing subject can be: the latest work on succinyl sulphathiazole and promin is naturally not included, although both compounds are mentioned. The tabular statements giving an order of preference for each of four drugs in a large number of different conditions (now 60) and of the frequency and import of various toxic manifestations, both of which we ventured to criticize as perhaps unduly dogmatic, are still included, and anthrax still figures mistakenly in the list of diseases for which sulphonamide compounds are of no value. The book is published in America by the Year Book Publishers, and in London by H. K. Lewis at 18s.

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THE NEW ERA IN INDUSTRIAL MEDICINE

The sound of shears, if not of axes, might be expected to be heard in the report of a Select Committee on National Expenditure, but such a report just issued, on health and welfare of women in war factories,[1] is by no means a bleak document. Those who compiled it have had obvious difficulty in keeping within their terms of reference—namely, the economic impact of problems of industrial health on war expenditure—and have touched on a number of social implications. So far from retrenchment, they make some bold and timely recommendations which will cost quite a lot of money. They urge, for example, the enlargement and strengthening of the medical branch of the factory inspectorate of the Ministry of Labour. The thirteen medical inspectors at present attached to the inspectorate are there to advise on occupational hazards and to carry out research and inquiries into specific problems. In the opinion of the Select Committee their number is not adequate to provide advice and instruction on a wide scale, nor have they the authority to secure the acceptance of their advice where it is most needed. Another recommendation is for the setting up of industrial health advisory committees, central and regional, to advise the Ministry of Labour, supply departments, regional boards, and employers on questions of industrial health, co-ordination of medical services, and the like. These would be channels in which information and advice would be conveyed down to the smallest factory. A central bureau is also proposed to keep factory doctors, nurses, and welfare staff abreast of recent knowledge and developments.

All this may well presage the opening of a new chapter in industry. Hitherto health services in industry, with all that they can effect in reducing the incidence of industrial disease, have depended principally upon arrangements made by firms who were progressive enough to realize their importance and large enough to justify the engagement of medical and nursing staff. No clear standards of industrial health have been worked out. Central direction has been lacking. The Ministry of Health does not include all health services in its brood, and this is one of the unincluded. The Ministry of Labour lacks the necessary organization to deal with these problems. At present there are only 150 whole-time and 582 part-time doctors employed in industry. In addition there are the examining factory surgeons, to the number of nearly 2,000, whose work is ordinarily that of examination of juveniles and inspection of processes. No doubt the work of these examining surgeons now includes some of the duties which fall to part-time works medical officers; but as there are over a quarter of a million factories in this country it will be realized that the amount of supervision which can be exercised by this small body of medical men can cover adequately only a fraction of the industrial population. Many defects are pointed out by the Select Committee, such as the lack of any close relationship between those who are working to maintain health inside the factories and those concerned with outside medical services—the works medical officers on the one hand and insurance practitioners, hospitals, and public health services on the other.

One important point is that the status, remuneration, and security of tenure are not such as to attract doctors into the field of industrial medicine. But the medical profession itself has recognized the need for a better industrial service. It was only in 1941 that the B.M.A. Committee's report on industrial health in factories was published, with its call for an extension of industrial medical service on both humanitarian and economic grounds, for more emphasis on the preventive aspects of industrial medical practice, and for closer association between the medical profession and industry. The Select Committee recommends that the Government should give the closest attention to the problem of securing a satisfactory industrial medical service, at least in all factories engaged on Government work, and in this connexion it considers that more use should be made of women doctors.

The circumstance which has led up to these proposals is the entrance on a large scale of women into industry. One and a half million women are now employed in industry who were not so employed in mid-1939. Large numbers of them have had no previous experience of factory life. A considerable proportion are married women, with their own special problems. Except in Royal Ordnance factories and certain private undertakings there has been no selective medical examination on entrance. Many women have been directed to work away from their homes. From various accounts these transfers have not always been carried out with much imagination. Women have been sent a considerable distance, and then it has been discovered that they are unfit, by reason of illness or mental disability, for the work to which they have been assigned—a thing which might sometimes have been obvious even to the lay official. Only the present scarcity of medical staff prevents the Select Committee from recommending that careful medical examination should be made in every case before a woman is transferred. The Select Committee makes a score of recommendations concerning hours of work, rest pauses, amenities, safety precautions—in 1941 the number of reportable accidents to women was 42,837, nearly three times as many as in 1940—and many other items in the management of personnel. It urges that plans be proceeded with for the early diagnosis of tuberculosis by mass radiography, that means of rehabilitation be provided for the injured worker, that visual standards be established for various factory processes, that attention be given to ventilation in factories, the more important in view of the black-out—often daytime black-out—conditions. It goes outside the factory and suggests that a pregnant woman should be eligible for unemployment benefit for two months before her confinement, as well as for a suitable period afterwards, to enable her to breast-feed her infant. Maternity benefit under National Health Insurance, whereby the mother receives £2, or a maximum of £4 if both she and her husband are insured, is insufficient, especially at such a time, when her earnings usually cease with her employment.

The entrance of women into industry has caused many misgivings. Women have special needs and are subject to special strains. Factory amenities that might be disregarded in the case of men assume a critical importance in the case of women, and the Committee even concerns itself with such questions as the colour of the walls in rest rooms—deprecating whitewash—and the provision of mirrors in wash rooms. What is more important—hours of work which are suitable for men may be too lengthy for women. We learn of one transport undertaking which began to employ conductresses under the same conditions as men, but within three months the women went on deputation to the management and said that they were unequal to the hours, and the schedules were revised and the pay

[1] *Third Report from the Select Committee on National Expenditure, Session 1942-3.* "Health and Welfare of Women in War Factories." H.M.S.O. (4d. net.)

readjusted accordingly. Another recommendation of the Committee is that women personnel managers and welfare officers should play an important part in selecting, interviewing, and allocating woman labour. "A man frequently does not appreciate the needs and difficulties peculiar to women." We hope that the new colour and pattern which the entrance of women into the factories is giving to industrial health and welfare organization will be permanently wrought into the industrial life of the future. The Select Committee says: "The State cannot afford to be other than a good employer." And if the State cannot afford it, neither can any private undertaking.

CIRRHOSIS OF THE LIVER

The term "cirrhosis" was introduced by Laennec for a condition which in many ways presents as enigmatic a problem as any other in medicine. There has consequently been no shortage of descriptive terms and attempts at classification: atrophic and hypertrophic cirrhosis, portal, biliary, and Hanot's cirrhosis, mono- and multi-lobular cirrhosis, and "hob-nail" or "gin-drinker's liver" have all had their vogue. Out of chaos an orderly system is now perhaps emerging which will mean something to the clinician. The tendency now is to recognize two main types—namely, portal and biliary (obstructive and infective). Hanot's cirrhosis is perhaps no longer a recognizable anatomical entity; healed yellow atrophy is being brought into line with cirrhosis. But we still seem to be far from a complete understanding of the aetiology and pathogenesis of cirrhosis. The cells of the liver are bathed in blood brought by the portal circulation and so are exposed to any noxious agent that travels along this well-worn route. According to the nature and extent of the injury a few may die; reaction may be severe or mild, or repeated, or slow and insidious; whatever the beginning, the end may be cirrhosis.

Moon,[1] in a comprehensive review, emphasizes the destruction of architectural pattern as a criterion of true portal cirrhosis, and states that of all the chemical agents used only a few have produced true portal cirrhosis—namely, carbon tetrachloride, phosphorus and alcohol together, manganese chloride and phenylhydrazine, and chloroform combined with infection. Chronic alcoholic poisoning is now almost off the list of cirrhogenic agents, and room on it is now being found for dietary factors, in either excess or deficiency. According to Earle and Victor[2] an excess of cystine in the diet produces fatty infiltration, portal haemorrhagic necrosis, a high mortality rate, and cirrhosis in all rats surviving more than two weeks. This cirrhosis is similar to that produced by selenium in rats and unlike that produced by some of the poisons mentioned above. These observations are in line with those of György and Goldblatt,[3] who found that in the hepatic injury of dietetic origin (necrosis and cirrhosis, with or without ascites) determining factors seem to be connected with the lipotrophic action of casein. Previous experiments[4] showed that focal or diffuse necrosis was observed in rats fed on a diet devoid of the vitamin-B complex, supplemented with aneurin, riboflavin, and pyridoxine. They believe that the lipotrophic action of casein is in direct proportion to its content of methionine and to the presence of choline in the diet, and that it is reversely influenced by *l*-cystine and supplements of fat. Relating this to human cirrhosis, they point out that a low intake of protein with insufficient supply of the vitamin-B complex (including choline) is a prominent feature of the diet of persons addicted to alcohol: it is as unnecessary to postulate an injurious effect of alcohol in cirrhosis as it is in the pellagra or beriberi of alcoholics. Some corroboration of the reported beneficial effect on cirrhosis in man of a highly nutritious diet supplemented with the vitamin-B complex comes from Patek and Post,[5] and from Lowry *et al.*[6] of the beneficial effect of choline, high-casein diet, or choline with a high-casein diet in rats with hepatic cirrhosis produced by low-choline and low-casein diet with added cystine. In Patek and Post's clinical trials after the onset of ascites, 72% of a treated as compared with 50% of an untreated series survived six months, and at the end of two years the percentages were respectively 45 and 21. There were also signs of bodily improvement and presumptive evidence of arrest of the disease. They suggest that there is an aetiological relationship—either direct or indirect—between nutritional deficiency and cirrhosis of the liver. Lack of essential factors might lead directly to the development of the disease, while the malnourished liver becomes more vulnerable to toxins. Spellberg *et al.*[7] produced cirrhosis of the liver in rabbits and fatty liver in guinea-pigs by feeding the animals on apparently adequate diets containing 20% of fat—chiefly butter fat—although some factor other than diet might have been responsible. It was recognized that the rabbit is not altogether satisfactory as an experimental animal because of the common occurrence of coccidial infection. In view of these results, those of Rao,[8] suggesting that the infantile cirrhosis of Hindu children (a variety of portal cirrhosis) is caused by a diet of cows' milk combined with *B. coli* infection, must be seriously considered. There is scope for further work along these lines. The study of spontaneous cirrhosis in animals in this field should not, however, be excluded, and reference might be made to the interlobular fibrosis in the liver of the normal adult pig, the more common occurrence of cirrhosis in the pig than in any other animal, and the fact that cirrhosis in the pig produced experimentally by carbon tetrachloride closely resembles the naturally occurring lesions.[9]

1 *Arch. Pathol.*, 1934, **18**, 381.
2 *J. exp. Med.*, 1941, **73**, 161.
3 Ibid., 1942, **75**, 355.
4 Ibid., 1939, **70**, 185.
5 *J. clin. Invest.*, 1941, **20**, 481.
6 *Publ. Hlth. Rep.*, Wash., 1941, **56**, 2216.
7 *Arch. Pathol.*, 1942, **33**, 204.
8 *Proc. Ind. Acad. Sc.*, 1941, **14**, 310.
9 White, E. C., *J. Path. Bact.*, 1939, **49**, 95.

THE PATHOGENESIS OF PEPTIC ULCER

A recent paper by Ask-Upmark[10] attempts to throw new light on the pathogenesis of peptic ulcer. Impressed by the frequency of peptic ulceration after all types of experimental damage to the liver, he contends that liver dysfunction is an essential factor in the aetiology. His experimental evidence consists of a study of post-mortem material and a series of tests of hepatic function in 26 cases of peptic ulcer. In 22 necropsies of hepatic cirrhosis he found 9 instances of chronic peptic ulcer. For his liver tests he used two varieties of the galactose-tolerance test, the Sjöström citric acid test, the Takata-Ara reaction, and the lipase determination of Rona. If all the tests are taken into consideration, 19 of the 26 cases of ulcer showed some abnormality of hepatic function.

The author considers that there is no evidence that the liver disturbance is secondary to abnormalities in the stomach, and he discusses at some length the mechanism whereby the liver might influence the stomach and duodenum. He favours the view that liver damage causes hypoglycaemia, which exerts an influence on gastric secretion. There is considerable evidence that hypoglycaemia does occur in peptic ulcer. The similarity of hunger pains and night pains to the symptoms of hypoglycaemia is quite striking, and the favourable effect of the administration of carbohydrates on the pain of peptic ulcer is also

10 *Acta med. scand.*, 1940, **103**, 280.

well recognized. The behaviour of the blood sugar in peptic ulcer has been studied by Christlieb,[11] who finds definite abnormalities in the glucose-tolerance test, the blood sugar rising too high in the hyperglycaemia phase and falling below normal limits in the hypoglycaemia phase. Ihre[12] has shown that insulin in the presence of hypoglycaemia is a powerful stimulant of gastric secretion, causing a rise in the pepsin concentration as well as hypersecretion. This is a vagal mechanism and can be abolished by cutting the vagi or by the use of atropine. Ask-Upmark shows that there is a relation between the liver and the central nervous system, citing as an example Wilson's disease (hepato-lenticular degeneration). He gives interesting case histories, where in one patient head injury, and in another encephalitis, were followed by gastric ulcer and evidence of liver damage. He concludes that the development of ulcer is mutually influenced by the liver and the central nervous system, but that probably the nervous influence is the more important. He visualizes the following chain of events: the hypothalamus, acting on a liver with impaired function, produces hypoglycaemia, which through a vagal reflex causes hypersecretion, this in turn leading to ulceration.

Most of us will find this chain about as difficult to visualize as the Indian rope trick. A more plausible explanation of the connexion between the liver and peptic ulcer has been provided by Cheney.[13] Superficial erosions develop in the gizzards of chicks fed on a diet which is deficient in a fat-soluble factor, occurring in association with, but definitely separate from, vitamin K. The same kind of lesion can also be produced by administering cinchophen to chicks, and the erosions become ulcers which closely resemble peptic ulcers in man. These cinchophen ulcers are more pronounced the more deficient the diet, and Cheney suggests that the ulcer-producing properties of cinchophen are due not to direct action on the lining of the stomach but to indirect effect on the liver. Damage to the liver may then interfere with the elaboration and storage of the anti-ulcer factor and its delivery to the stomach wall. The distribution of the "anti-gizzard erosion factor" is wide, and a direct deficiency in human diet is unlikely, even under conditions of war; but the possibility of a deficiency conditioned by a disturbance of the liver is well worth bearing in mind at the present time, when gastritis and peptic ulcer present such a formidable problem, and when potential liver poisons are widely used in industry and in the manufacture of munitions.

[11] *Dtsch. Arch. klin. Med.*, 1938, **181**, 394.
[12] *Acta med. scand.*, 1938, Suppl., 95.
[13] *Arch. intern. Med.*, 1942, **70**, 532.

STARVATION IN SOUTHERN FRANCE

A moving plea that the blockade be lifted to allow 3,000 tons of food to be sent to Marseilles to preserve for another year the lives of 100,000 children in Southern France was made at a recent conference in London by Mr. Howard E. Kershner, director of relief of the American Friends Service Committee. Mr. Kershner spoke from personal experience in France of feeding French children since the Franco-German armistice. An acute food shortage was produced in Southern France in 1940 by the flood of refugees which in some towns multiplied the normal population by ten, and there had been no recovery. Failure of local production was due to absence of farm labour, enforced slaughter of farm animals, and shortage of fertilizers and seed. In this part of France, Mr. Kershner declared, three-fourths of the children were losing weight. Children who should have gained 4 to 5 cm. in height had gained only 1 to 2 cm.; often a gain in height was accompanied by loss of weight. Athletics had been given up in the schools, where the children sat about too weak to play. Newborn babies were usually about two-thirds of the normal weight; weights of infants had fallen to little more than one-half, and infant mortality had climbed up to three times the normal. He had seen grown men who weighed no more than 75 lb.

For two years his Service Committee had supplemented for 100,000 children the meagre French ration, on which it was scarcely possible to keep alive. This supplementary food had not come through the blockade: it had been accumulated previously from various sources in Central Europe, Africa, and Asia, even as far away as Indo-China; but these doors were now closed. A milk distribution scheme was organized, and it was remarkable to see how the weights of infants, which had been steadily decreasing, flattened out as soon as the milk distribution was made, and presently began to ascend; but the United States Government had refused permission for payment to be made for additional supplies of powdered milk which it had been arranged to buy in Switzerland.

Mr. Kershner, meeting an obvious objection, said that in his experience not a mouthful of food had been lost to the Germans, who had not interfered with the relief operations. Germany to-day was not taking as much food from France as she was entitled to take under the terms of the armistice, for the reason that the food did not exist in sufficient quantities to make it economically possible to take it away. If the British and American Governments would allow this extra food to enter the country it could be sent over in neutral ships (Swedish or Portuguese), the International Red Cross was willing to accept the responsibility, and 140 non-American workers in Southern France would feed the children. The food was available in abundance in America. In the interests of shortening the war and making reconstruction less difficult, he urged that the necessary facilities to save the life of the children of starving Europe should be granted.

TREATMENT OF DIABETIC COMA

Joslin and his co-workers[1] have recently reviewed their results in the treatment of diabetic coma since 1923. Since that time 525 patients have been treated, with a total mortality of 11%, and, since 1940, 62 consecutive cases without a single death. These striking results deserve close study. The American workers find that the commonest cause of coma is laxity in treatment either through dietary indiscretions or through failure to take the proper amount of insulin; the next is infection; in a third group coma is the first recognized sign of diabetes. Death cannot be correlated with any single symptom or sign, and the prognosis can be assessed only by the consideration of many factors, particularly age, degree of shock, blood pressure, depth of acidosis, blood sugar, plasma carbon dioxide, and blood non-protein nitrogen. Of these the single most important consideration is the depth and duration of unconsciousness before the institution of treatment.

The treatment of diabetic coma is essentially a hospital procedure, and preparations must be based on the realization that it is an acute medical emergency for which early, prompt, and vigorous treatment is absolutely essential. The medical, nursing, and laboratory staff should be organized to this end. As coma is due primarily to insulin insufficiency, adequate insulin dosage is the first consideration, and the first 50 units should often be given at home or on the way to hospital. On arrival the first dose should be between 20 and 100 units subcutaneously, and if the patient is unconscious and shocked a similar dose may be

[1] *J. Amer. med. Ass.*, 1942, **119**, 1160.

given intravenously. Further doses are then given half-hourly or hourly, the average patient needing at least 200 units in the first two or three hours. Subsequent dosage depends on the initial blood sugar response. If this is slight, larger doses of insulin should be administered more frequently, and it may be necessary to give 100 or more units at half-hourly or hourly intervals. The level of the blood sugar, the urinary sugar, and the clinical conditions—especially the mental state and the type of breathing—are the most valuable guides.

Next in importance in treatment is the administration of fluids and salt, and isotonic sodium chloride solution, either intravenously or subcutaneously, should be begun at once, four to five litres in the first 24 hours being an average amount. Relatively more should be given by the mouth as recovery takes place. Other important measures include gastric lavage (which should be performed carefully on admission in almost every case), enemas, and small feeds such as broth, gruel, or tea with sugar by the mouth after about the first six hours. Dextrose is contra-indicated during the first few hours of treatment because there is already excess in the body fluids, and to give more masks the response to insulin. Alkalis are also considered unnecessary. Patients with prolonged unconsciousness and peripheral circulatory failure with very low blood pressure respond best to large doses of insulin and continuous saline infusions, with additional transfusion of plasma or whole blood in selected cases.

In summing up, Joslin and his colleagues emphasize that coma should be prevented, and that the occurrence both of coma and of death due to it shows that someone has blundered. The key to success lies in the education of the diabetic patient and of his doctor. These conclusions and their application are of profound importance. However good the actual technique of treatment in hospital or clinic, the incidence and mortality of diabetic coma will not be appreciably reduced until every diabetic patient is taught how to look after himself and when to seek further medical advice, and until every doctor knows how to manage diabetes in the home. In this country, especially in rural districts, delay in making the diagnosis and getting the patient to hospital is sometimes so considerable that the best treatment comes too late. Diabetic coma is as grave and as dangerous an emergency as an acute abdomen, and must always be treated as such.

HELP FOR THE SHIPWRECKED

To be cast adrift in a small boat on the open sea with scanty provisions and only a hope of being picked up is a situation the obvious drama of which has always made a powerful appeal to the imagination. To be so closely confined within such a vast space, to be exposed to every extremity of climate night and day, to sail upon water in an agony of thirst, to watch the effect of all this upon one's companions, and to endure, must call in the highest degree imaginable upon those mental and moral resources which constitute the heroic spirit of man. In an island country this is an eternal theme, and any measure or effort that will help to maintain the lives of those that "go down to the sea in ships" will, in time of war especially, command the heartfelt support of all men and women. Although chance plays its haphazard part in survival from shipwreck, it can be helped or hindered, or even destroyed, by the conduct of those who escape immediate death by drowning, and especially by the behaviour of the man in charge of a lifeboat's company. This conduct must be based on principles of psychology and physiology. A hundred and fifty years ago a seaman's guide was published which on the basis of experience offered advice to the shipwrecked. "Hope, perseverance, and subordination should form the seaman's great Creed and Duty, as they tend to banish despair, encourage confidence, and secure preservation." This and other excerpts from the guide are given in another manual just published and entitled "A Guide to the Preservation of Life at Sea after Shipwreck."[1] It has been prepared by a special committee of the Medical Research Council and is yet one more practical witness to the imagination and flexibility of this institution, to which the medical profession is so profoundly indebted for its many and sterling services to this country in time of war. A full abstract of this valuable document is printed elsewhere in this issue.

B.C.G. VACCINATION

Little is heard to-day about B.C.G. vaccination; reports of its use in other countries reach us with difficulty or not at all. Yet methods of controlling tuberculosis are not so numerous or effective as to justify completely disregarding it, particularly when the incidence of the disease is rising. B.C.G. vaccination is extensively used in Russia: it is used to an increasing extent in certain parts of the United States; and in Sweden it is compulsory in nurses who are tuberculin-negative. Wallgren has recently reviewed[2] his experience in Göteborg, Sweden, a town with a population of 250,000, where some 1,069 persons, chiefly children, in contact with tuberculosis were vaccinated by the intradermal method from 1927 to 1937. Great care was taken to vaccinate only non-infected people by separating them from contact for at least six weeks and testing with tuberculin. Most children were also kept away from contact for two months after vaccination. There was a follow-up of the vaccinated children in 1938, and it was found that of 397 vaccinated soon after birth who subsequently remained in contact with tuberculosis none died of the disease, and in only three were mild tuberculous lesions detected later. The parents of only ten children refused vaccination, but of these ten who were not vaccinated five died of tuberculosis, and at least one other developed tuberculous disease. Wallgren rightly emphasizes other favourable factors associated with the vaccination—for instance, the period of separation from contact and the special care that may be given by relatives to these children afterwards. Discussing immunity against tuberculosis, he points out that, as in syphilis, specific immunity may protect against fresh tuberculous infection but not necessarily against the virus already present in the body. Similarly, the relative immunity acquired as the result of B.C.G. vaccination might lessen the severity of the primary lesions, but would not necessarily prevent chronic dissemination or progressive pulmonary tuberculosis later. If, however, it is true that mild primary tuberculosis in adolescence is less likely than extensive primary lesions to be followed by progressive pulmonary tuberculosis, B.C.G. vaccination might also be expected to lessen the risk of post-primary tuberculosis in young adults. In this connexion Wallgren recalls, as was also pointed out by Kayne[3] and Pagel[4] recently in this country, that progressive pulmonary tuberculosis in adolescence and young adult life may be a fairly recent sequel to primary infection. From this he concludes that there are good grounds for recommending B.C.G. vaccination of young people before leaving school and before they go out into the world; this is already the practice in a few Swedish towns. Finally Wallgren produces evidence in support of his thesis that the protection obtained from B.C.G. vaccination remains much longer than is commonly believed—often ten years and even more.

1 M.R.C. War Memorandum No. 8. H.M.S.O., 1943. (4d. net.)
2 *Arch. Kinderheilk.*, 1941, 122, 65.
3 *Proc. roy. Soc. Med.*, 1942, 35, 483.
4 Ibid., p. 489.

HELP FOR THE SHIPWRECKED: A PRACTICAL GUIDE

As announced on another page, the Medical Research Council has just issued "A Guide to the Preservation of Life at Sea after Shipwreck." The information given in this guide summarizes the pooled knowledge of sailors and scientists, and it is hoped that the pamphlet will be studied by officers of the Royal Navy and the Merchant Navy and by medical men. The members of the committee drawing up the guide were Surg. Vice-Admiral Sir Sheldon Dudley, Surg. Rear-Admiral G. Gordon-Taylor, Surg. Capt. M. Critchley, Prof. A. N. Drury, Surg. Cmdr. R. A. Graff, Sir Thomas Lewis, Dr. R. A. McCance, Air Commodore T. McClurkin, Squad. Leader T. F. Macrae, D.Sc., Dr. Spencer Nairne, Capt. R. Oliver-Bellasis, R.N., and Dr. B. S. Platt. The committee was appointed in October, 1941, at the request of the Medical Department of the Navy, with the following terms of reference: "To examine the conditions to which Naval and R.A.F. personnel may be exposed after loss of ships or aircraft, when they may be adrift in boats or rafts or isolated in the desert; to examine the physiological aspects of those conditions, with special reference to water requirements, exposure to extremes of heat and cold, food requirements and stimulants, and to frame practical recommendations." In the preparation of the pamphlet the committee makes special acknowledgment to Cmdr. N. D. Holbrook, V.C., R.N., and Cmdr. J. Quayle, R.D., R.N.R.

Almost everything, the introduction states, depends on the bearing and conduct of the officer in charge; experience has shown that comfort and chance of survival depend upon the frame of mind of the boat's company. The store of vital energy possessed by each member of the lifeboat's company should not be wasted by useless exertion or the development of a "bad frame of mind." The contents of the pamphlet are then set forth under the following main headings: "Routine Preparations and Precautions in the Ship"; "Action in Emergency"; "Abandoning Ship"; "Procedure in Lifeboat"; "The Treatment of Sick and Wounded"; "Lifeboat Ailments"; "Steps towards Rescue"; "Treatment after Rescue." The points of likely interest to medical men are summarized below.

Some Anticipations

First-aid outfits, if not hermetically sealed, should be inspected frequently; damp articles must be dried or replaced.

If bomb hits are expected and duties permit, the effect of blast or of fragments and the risk of being thrown may be minimized by lying flat on the deck with head and body hard against a ledge-side or deck. Some protection is afforded if one stands with the head against something solid. If there is risk of flash-burns from explosions the skin should if possible be covered, even though the clothing be only thin.

The shipwrecked have often suffered unnecessarily from lack of clothing. Therefore, in abandoning ship, waterproof and any extra warm clothing—such as blankets or Duffel coats—should be gathered up. Too much clothing is better than too little; it will protect against the sun as well as against the cold. A pair of dry socks should be carried in the pocket or in a waterproof package. Modern lifebelts, it is pointed out, will support a person in the water fully dressed and wearing sea boots. Every effort should be made to take extra water: "*Water is more important than food.*" Containers must be improvised. Water bottles or similar containers should be filled. "Masters who have appreciated the paramount importance of an adequate water supply have succeeded in improvising stowage for as much as 120 gallons per boat." It is not likely that more food will be needed than is stored in the lifeboat.

Going Over the Side

If a man has to go over the side of the ship, he is advised to go over the lower side if the ship has listed. Going over the upper side carries with it the risk of being hurt by barnacles and marine growths and of fracturing the ankles against the bilge keel. If the lower side of the listed vessel is the weather side, a man should jump into the water from the bow or the stern, whichever is the lower, so as to avoid being washed back on board. He should jump into the water feet first, and not dive. If the sea is covered with fuel oil he should hold his nose when jumping, and when swimming through a patch of oil should keep head and eyes high and mouth closed. The effects of contact with oil discharged into the sea are not, however, serious. Swallowed oil may make a man sick, and if it gets into the eyes cause temporary blindness. If a man has to jump from a ship into burning oil, he may, if a good swimmer, avoid being burnt. He should jump feet first into the flames, swim as long as possible under water, then spring above the flames to breathe, taking a breast stroke to push them away; then back under the water again. Men have been able to traverse 200 yards of burning oil in this way. It is evident that the lifebelt and other cumbersome clothing should be removed to make it possible to do this.

Many men, it has been pointed out, have been drowned through panic action and thrashing about in the water. A man should look for a lifeboat, raft, or other floating object, and swim slowly towards it; he should not exhaust his strength by swimming about uselessly. Swimming or floating on the back lessens the danger of injury from under-water explosion.

Adrift

Energy must be conserved in the lifeboat. Singing and shouting, for example, use up strength, and cause loss of valuable water in the breath. Each man, unless badly wounded or exhausted, should have a job to do, however small. All wet clothing should be squeezed as dry as possible, but clothes should not be taken off for this purpose unless the weather is warm and dry and the wind moderate. Special attention must be paid to the feet. Wet socks and boots should be taken off and dry socks put on—and the boots too, when dry enough. If there is risk of "immersion foot," then boots are better removed from the start if the feet can be well covered with socks, wrapping, and waterproof clothing; boots should be left on if the feet cannot be kept dry, but must be removed once swelling of the feet is noticed.

Protection must be afforded against cold winds, rain, and spray, and against sun and heat, by means of canvas or wooden screens or an awning. In the heat too much clothing should not be taken off, as it protects the skin against sunburn. Legs and feet should be kept covered or in the shade. The wearing of a shirt or vest and headgear kept wet by occasional soaking in sea-water will afford some protection against tropical sun. Fuel oil, and greasy preparations such as ointment, tend to "fry" the skin and should not be used against sunburn. The glare of sun on the water may be minimized by tying a cloth or bandage over the nose. Bathing once or twice a day is allowed provided that no sharks or other biting fish are about, nor any stinging jellyfish nor Portuguese men-of-war. Sharks apparently are not as dangerous as reputed, and when they rub themselves against the lifeboat are more often than not trying to rid themselves of sea-lice.

Water and Food

The officer in charge must plan the rationing of water from the start. The size of the ration depends upon the likelihood of being picked up. If this is not likely to happen for a week, then no water need be given during the first 24 hours unless the men have been sweating from hot weather or exhaustion. The ration recommended is 18 oz. per man daily, which should be continued until there is one pint per man in hand; the ration must then be reduced to 2 oz. a day. It is pointed out that it is foolish and even dangerous to ration water on, say, a 60-day basis when there is a good chance of being picked up in 10 days. The experience of survivors suggests that moistening the lips and rinsing the mouth out with sea water are helpful, and also that small quantities may be used for soaking biscuits. "There is, however, no doubt that large draughts of sea-water cause death, and even small amounts may prejudice a man's chances of survival on a long voyage. Permission to *drink* sea-water, therefore, should never be given." Rectal administration of sea-water does not relieve thirst and is harmful. It is recommended that every drop of rain-water should be collected, and that no tin or vessel which can hold water should be thrown away. Pools of water formed of melted ice on ice-floes, provided they are not brackish from salt-water spray, may be drunk. The drinking of urine is harmful and should be forbidden.

If water rations are low, the food rations should consist mainly of fats, starches, and sugars, and should contain only small amounts of protein. The quantity of salt included in the

food should also be small. Fats, besides providing concentrated energy, allay hunger better than other foods, and for this reason a biscuit with a high fat content is recommended. Pure butter-fat is the best kind of fat, and keeps well in airtight containers even in tropical climates. Foods rich in protein increase the body's requirements of water and should be avoided. Spirits have no thirst-quenching value and may be dangerous. If available they can be given to severely wounded men to secure sleep and ease pain. It is pointed out that owing to the small amount of food eaten the bowels will probably not act, and that the quantities of urine passed will diminish, but that this need not cause alarm.

Keeping Awake

Between watches it is important to get regular sleep. When fatigue increases, the man at the tiller or on watch may find it difficult to keep awake. He should then use the "energy tablets" provided—methedrine or amphetamine (benzedrine). If it appears that the whole party may be overtaken with severe exhaustion, it is considered better that all should keep awake, and that "energy tablets" should be issued. They are said to "(1) lessen feelings of fatigue and exhaustion, promote alertness, raise the spirits, and prolong the will to 'hang on' and live; (2) prevent sleep." Methedrine or amphetamine should be given when men are beginning to be worn down by hardship. If the shipwrecked are in Carley floats, rafts, or dinghies, and conditions are severe from the outset, tablets should be given at once—two at sunrise, at midday, and at sunset. If the conditions improve the tablets should be stopped. It may be dangerous to drop off to sleep in a Carley float, as the risk of drowning is increased. As a lifeboat party must be prepared to hold out for long periods, the tablets should not be used in the first few days. Single doses of two tablets may be given for special purposes—for example, to the man at the tiller, or when a special effort is necessary. If the whole party is overcome by exhaustion each man is given two tablets at sunrise and sundown. They should not be given to the wounded, to the excitable or hysterical, or to those whose minds are wandering.

A list is given of the first-aid equipment, and details of the Schäfer method of artificial respiration. There are also practical instructions on the emergency treatment of wounds, broken bones, burns and scalds, and shock. Among the short practical hints for the treatment of lifeboat ailments, dangling the hands in the sea is recommended as an aid to urination when this is felt to be difficult.

Frostbite

Two conditions are treated at greater length—namely, frostbite and "immersion foot." Frostbite at sea is rare unless the sea water is freezing. The skin, and sometimes deeper tissues, become actually frozen, the damage being caused by minute crystals of ice forming within the skin, which turns pale and yellowish-white, looks opaque, and feels of "wooden hardness." The actual freezing occurs suddenly and may pass unnoticed, as will the spread of the frozen patch. On thawing, the skin becomes inflamed and blistered. After severe freezing the part frozen dies and comes away after "blackening." Frostbite of the face may be detected by wrinkling it up to detect areas of stiffness. Small frozen areas may be thawed rapidly by covering them with a warm hand. Larger areas severely frostbitten, such as a whole foot, should be thawed gently by placing it inside a companion's clothes. Frost-bitten skin should not be rubbed with snow or otherwise. In the treatment of frostbite in a rescued person the thawed part should be put in cold, not warm, water, the rest of the patient being warmly wrapped up. On thawing, if the pain is severe, the part may be cooled for a short time with cold water, ice, or snow; and after thawing warming up must be gradual. The limb must not be warmed in front of a fire or by the application of hot water or bottles. The affected part should be cleaned and dried gently, dusted with sulphanilamide powder, wrapped in clean material, and kept at absolute rest.

Immersion Foot

"Immersion foot" is different from frostbite. The term is applied to damage of tissues immersed many hours in cold water or mud. The limb immersed is painful, red, numb, and weak. Numbness and weakness increase, and the limb swells. If immersion ends and the limb is warmed, early signs of damage quickly disappear. If the immersion lasts for days, damage will be more serious and blisters or dark patches appear, and the skin become broken. The damage, it is pointed out, comes from cold, and the chief source of cold in boats is cold water, out of which the feet should be kept if possible; also, if possible, boots should be emptied and socks dried. Feet and toes should be moved frequently. If the feet are swollen, numb, or tender, rubbing the skin will do more harm than good. In the treatment of immersion foot in a rescued man the affected limb should be kept cool, the rest of the body being heated. The limb should be warmed only gradually, and heating or rubbing it in any way is harmful. The temperature of the skin should be reduced to about 80° F., but not much below 70° F.; it can be taken by placing a bath thermometer against the skin under a covering of cotton-wool.

Correspondence

Surgery of Oesophageal Cancer

SIR,—In your annotation (Jan. 9, p. 48) you very kindly refer to my case of successful removal of the thoracic oesophagus by the "pull-through" method. But it would be unfortunate if this plan was regarded as purely a feat of prestidigitation, for it was deliberately planned after most careful consideration and prolonged study of the anatomical and pathological features involved, so that in no sense could it be described as "a trick." Although at the present time I regard the transthoracic operation as the best for dealing with growths of the mid-part of the gullet, I am not prepared to say that the "pull-through" method will eventually be entirely superseded. In all operations I like to work under the direct guidance of the eye, but none the less it must be admitted that sometimes blind procedures, like suprapubic enucleation of the prostate, are singularly successful. It is significant that the "pull-through" method has proved successful in the hands of other surgeons, and I would refer you to a well-illustrated account of the method together with the record of a successful case by Prof. A. W. Fischer from the University Clinic at Gieszen (*Arch. klin. Chir. Sonderabdruck*, 1937, ans. 189). In a personal communication I was also informed that a second successful operation had been carried out by one of Prof. Fischer's assistants. Unfortunately patients suffering from this disease still reach the surgeon when the condition has advanced beyond the wall of the oesophagus, which has consequently become fixed by infiltration to some of the surrounding structures; or there is involvement of the lymph nodes. The most important thing for the profession to realize is the necessity for thorough investigation of all cases of dysphagia at the outset. In this way a delay of many weeks could be avoided, and it might even be possible for a fair proportion of such cases to reach the hands of the surgeon while the disease is still limited to the walls of the gullet.

The surgery of malignant disease of the oesophagus is still in the experimental stage and there are many unsolved problems in connexion with its development, but whatever method of approach is to be employed the earlier it can be undertaken the better the chance of success.—I am, etc.,

G. GREY TURNER.

Filaria Worm in Eye

SIR,—I was very interested in Squadron Leader M. C. T. Reilly's note (Jan. 23, p. 103) on the removal of a filaria from the eye. Since 1937 I have seen two cases in the ocular adnexa, both patients having returned from abroad a few years previously. One was found in the upper lid and seized with forceps. Some novocain was then injected and the worm removed through a small incision. Apparently injection of novocain as a preliminary sends the worm post-haste in the opposite direction. The other was removed through the subconjunctival tissues in a similar manner to that described by Squadron Leader Reilly. Both were *Filaria loa*.

Duke-Elder has an excellent chapter in Volume III of his *Textbook of Ophthalmology* and quotes various cases dating from 1771 in which filaria have been found in almost every

tissue of the eye—anterior chamber, iris, lens, vitreous, and retina—most of which, however, appear to relate to *Filaria bancrofti*, with successful removal in many cases. When the worm is seen in the anterior chamber, a keratome incision is made without loss of aqueous, and as the worm approaches the incision the lower lid is depressed and the worm floats out in the escaping aqueous. When inside the eye, the filaria may be tolerated for some time, but eventually considerable inflammatory reaction takes place, the majority of cases resulting in severe irido-cyclitis associated with keratitis. Duke-Elder makes the very interesting observation that intra-ocular filarial parasites are more common in animals than in man, and they have been found in horses, oxen, dogs, and even in frogs and fish.—I am, etc.,

Edinburgh. C. R. DUNCAN LEEDS.

Orbicularis Oris in Facial Paralysis

SIR,—The subject of this letter may at first sight appear to be, but in reality is not, trivial. In a certain proportion of lower motor neurone facial paralyses there is almost complete recovery with the exception of a disfiguring retraction at the angle of the mouth on the affected side. For years one attributed this to contracture in certain muscles which had previously been the subject of flaccid paralysis, a phenomenon for which one was unable to find any analogy elsewhere. (The shortening of the muscles on the back of the forearm which sometimes follows a "drop" wrist would not be analogous.) It is now a good many years since I came to the conclusion—as no doubt other observers also have—that this facial deformity is due to non-recovery of the orbicularis oris, and consequent lack of opposition to the tone at rest, and activity in action, of the muscles which retract the angle of the mouth and whose motor nerve fibres have recovered. This constitutes a real disfigurement, and, in some, considerable psychological disturbance, and my object in writing is to suggest that it would seem to justify in itself the operation of decompression, which is hardly a major one, in the hope that the fibres destined for the orbicularis oris, possibly peculiarly exposed to pressure in the canal, may thereby be released.—I am, etc.,

London, W.1. ROBERT STANTON WOODS.

Phosphorus Poisoning from German Bullets

SIR,—Apart from drawing the attention of the profession to the possibility of fatal poisoning from the new types of German bullet, my main object in publishing my case (Dec. 5, p. 664) was in the hope of eliciting suggestions for treatment. The chief obstacle in eliminating the phosphorus or in rendering it innocuous before it gets absorbed into the circulation is the difficulty of reaching it, because the effect of the explosive charge scatters and spreads the phosphorus forcibly and deeply into the surrounding tissues. This deep infiltration would not occur, of course, in the case of the non-explosive type of phosphorus bullet described by Dr. J. R. F. Popplewell (Jan. 23, p. 111).

I have had communications from various sources, including such a high authority on toxicology as Dr. Roche Lynch, and I gather that there are only three commonly recognized chemical antidotes—i.e., copper sulphate, potassium permanganate, and hydrogen peroxide. A disadvantage of copper sulphate is that it is a poison itself, and is also locally deleterious to the tissue; further it would probably only act on the phosphorus lying in the superficial layer of the wound. Potassium permanganate 2% or hydrogen peroxide (half-strength) should cause no appreciable damage to the tissues, would be likely to penetrate more deeply (especially the hydrogen peroxide on account of its physical action), and could be applied continuously without detriment. The increasing difficulty in obtaining supplies of potassium permanganate and the expense in using large quantities point to hydrogen peroxide as the antidote of choice.

In treating a case of bullet wound the track of which is found to emit a phosphorus-smelling vapour, I recommend: (1) thorough opening up of the track, with extensive excision of all tissue practicable which shows any sign of damage; (2) irrigation of the wound with hydrogen peroxide—a weak solution at first for fear of a too violent reaction; (3) leaving the wound widely open and lightly packed with gauze, with Carrel's tubes placed in its depth; (4) continuous drip irrigation (or half-hourly irrigation) through these tubes with hydrogen peroxide half-strength; (5) administration of glucose, preferably by continuous intravenous drip, in order to assist the liver cells before they become irretrievably damaged.

In reply to Dr. R. E. Rewell's criticism (Jan. 9, p. 56) as to the incompatibility of having a high blood urea in the presence of liver necrosis, I would emphasize that the presence of these two conditions at the same time was an undoubted fact in my case. Facts may be stranger than fiction, and theoretical considerations must give way or be modified if they do not fit in with them. A scanty urine is known to be a phenomenon in acute phosphorus poisoning. Although the kidney damage does not appear to be great compared with that in the liver, the renal function may be impaired prior to the hepatic, and, if so, this might be an explanation of the high blood urea.—I am, etc.,

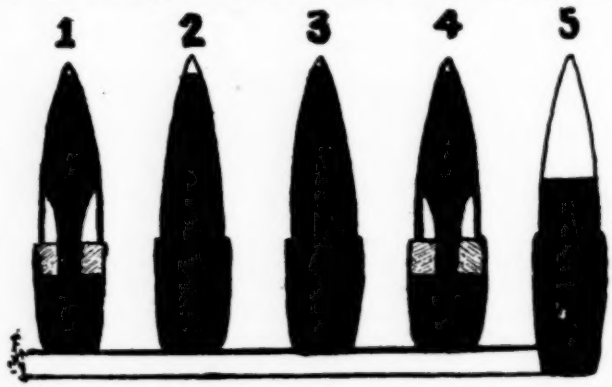
Norwich. A. J. BLAXLAND.

German Explosive Bullets

SIR,—Dr. J. R. F. Popplewell in his letter (Jan. 23, p. 111) is to be congratulated for bringing some details of German explosive bullets to the notice of the medical profession, and for having had this ammunition examined, which procedure is not entirely devoid of personal risk. That the enemy was making use of a variety of types of ammunition in his aircraft has, of course, been known to the authorities of all three Services since the early stages of the present hostilities, although, as Dr. Popplewell correctly observes, "the medical profession as a whole were kept in the dark" on this matter. I do not know why.

The following additional details on this subject may be of interest to the medical profession generally, and assist them in their treatment of victims from enemy machine-gun fire. Early in 1940 I obtained a sample of ammunition taken from the machine-gun belt of a captured Messerschmitt, and on this I carried out an *x*-ray examination. It was found that the enemy was using five different types of ammunition arranged consecutively in the following order: (1) ball ammunition; (2) armour-piercing; (3) armour-piercing with tracer; (4) armour-piercing with incendiary; (5) explosive bullet.

The internal structure of all five types of these bullets was shown on the radiograph, but there appeared to be little difference in the internal structure of the ball (No. 1) and armour-piercing incendiary (No. 4), and also a similarity between armour-piercing (No. 2) and armour-piercing tracer (No. 3). The explosive bullet, however, is distinctive in having



a semi-opaque distal half of the bullet, while the basal portion exceeds the length of any of the other four types by 0.3 cm. All five types of ammunition were, of course, sectioned and examined by an expert armourer, and the different types corroborated. The illustration above is a diagrammatic representation of the radiograph.

If an explosive bullet became lodged somewhere in the body and had failed to go off, a potential danger would exist to both the patient and the medical attendant. It might therefore be a wise precaution, in the interests of all, to have these casualties resulting from enemy machine-gun fire *x*-rayed before they are operated on, but, of course, excepting first-aid treatment.

It is just possible, in view of the above investigations, that the appearance of an explosive bullet would be determined by *x*-ray examination if the bullet was not too deeply seated in the tissues so that the appearance of the semi-opaque distal half became masked. I think the widest publicity of facts

such as these should be made in the interests of the profession, apart from the help that this might render in the treatment of the patient.

In view of these observations, I should be interested to know if any medical practitioners in the country have been able to determine from an *x*-ray examination the nature of a bullet lodged in the body of a patient whom they have been treating.—I am, etc.,

London, W.1. NORMAN P. HENDERSON.

SIR,—In his letter under the heading "German Explosive Bullets" (Jan. 23, p. 111) Dr. J. R. F. Popplewell describes and depicts two types of the .312 Mauser bullets as used by the enemy in aircraft automatic guns. Type 1 is admittedly an explosive (and expansive) bullet in view of the soft nose and the detonating charge in its base; but Type 2 would appear to be incorrectly described as an explosive bullet. Is it not rather of the "tracer," "incendiary," and "anti-petrol-tank" variety?

If, as your correspondent intimates, many members of the public have come into possession of these projectiles they will do well to hand them over to the military authorities. Fulminate detonators are apt to explode to a slight shock, such as a fall off the mantelpiece, and the yellow or white phosphorus, apart from its toxic nature, is highly inflammable and liable to ignite should the oxygen of the air gain access to it.—I am, etc.,

CHARLES M. BEADNELL,
Towyn, N. Wales. Surgeon Rear-Admiral.

Pasteurization of Milk

SIR,—During my work over many years I have been greatly impressed with the diseases and, in some cases, fatalities due to the drinking of raw milk. These diseases have included not only tuberculosis but also a considerable number of *Brucella abortus* infections which have frequently been unrecognized. My experience is that undulant fever in its various forms is much more prevalent than is commonly supposed. I have, therefore, been strongly in favour of pasteurization to safeguard the health of the community. In my own household the rule has been that milk should be boiled, but it is impossible to arrange that members of one's family should not drink raw milk outside their home. One of these developed a severe form of undulant fever lasting well over a year.

It is very easy to talk about the production of clean milk, but a very different thing to get it to the market and to the public in a clean and safe condition. Being much interested in this problem, I decided in 1939 to try to produce clean milk myself, and learn at first hand the difficulties. With this object in view I built a model cow-shed, which has every facility for complete cleanliness, and which is cleaned throughout twice a day and washed out with disinfectant weekly. I then imported from Guernsey, where there is no tuberculosis among the cattle, cows which had passed the tuberculin tests, and also the agglutination tests for *Brucella abortus*.

Every conceivable care with regard to clean milking has been scrupulously carried out, including regular washing of udders and teats with disinfectants, clipping of udders, tying of tails, washing and disinfecting milkers' hands between each cow, etc. The milk is cooled immediately each cow is milked, and all utensils washed, scrubbed, and sterilized twice daily. In spite of every precaution *Brucella abortus* infection broke out among my cattle. How this can be avoided I do not know. This disease is extremely prevalent among cows, and I feel it is quite probable that the infection may be brought to the pastures by birds. I do not believe that this disease can be easily prevented. The cows have been tested with tuberculin at regular intervals and there are no reactions. The milk, after it has left my dairy, has to be taken to a roadside spot, where it stands until it is collected by an open lorry, which travels round the neighbourhood and then takes it to a collecting depot, whence it is sent to London. During this transit, especially in the summer when the sun is shining on the churns, the milk must be warmed up again. It is collected about 10.30 a.m.

I have had bacteriological examinations of freshly drawn milk made by Dr. Fraser at the Bristol Royal Infirmary which are satisfactory, and of which the following is an example: "Plate culture at 37° C. 170 colonies in 1 c.cm.; plate culture at 20° C. no colonies in 1 c.cm.. No *B. coli* in 10 c.cm. The deposit shows a very rare leucocyte; no organisms seen. The bacteria present are scanty and chiefly *Staphylococcus albus*." But in spite of this and every precaution the mixed night and morning samples have not been satisfactory, the methylene-blue test has not been satisfied, and *B. coli* have been present in various analyses.

It is impossible in any cow-shed to prevent a certain amount of dust, carrying bacteria and floating in the air, which may easily contaminate the milk with *B. coli*, while flies in summer may be dangerous. It must be remembered that cows are by nature particularly unclean animals with their splashing excreta. The chief sanitary inspector of the county describes my quarters as excellent, and the dairy experts find no fault with technique. As the result of this experiment I am more than ever convinced that there is only one way to safeguard the community, which is to pasteurize the whole of the country's milk.—I am, etc.,

Churchill, Somerset. J. R. CHARLES.

Diphtheria during Immunization

SIR,—Dr. G. A. Garrett (Jan. 23, p. 102) described a case of a type which has great significance in any evaluation of diphtheria immunization. By referring to it as one of diphtheria, Dr. Garrett has assumed that a positive swab and a sore throat are synonymous with diphtheria. From his very concise account of the clinical evidence and the rapid response to anti-streptococcal chemotherapy it is quite clear that he was dealing with a case of follicular tonsillitis, and the positive K.L.B. swab was a coincidence of no pathological significance. Since immunization has been more extensively practised such a set of circumstances is not uncommon, as medical officers of health and isolation hospital superintendents well know.

Immunization protects against the disease and not against a positive swab, and, just as a typhoid carrier can contract enteritis from some other cause, there is no reason why a carrier of K.L.B. should not suffer from a streptococcal or other form of throat infection. If we are to gain anything like an accurate estimate of the effect of immunization on the *disease* diphtheria it is imperative that medical officers of health should carefully investigate every case, whether immunized or not, which is notified as diphtheria, particularly when the decision to notify rests on a positive swab. Since for all practical purposes all such cases are removed to isolation hospital this should not offer any undue difficulty.

The general practitioner, even if he doubts the diagnosis, quite rightly cannot ignore a positive swab, but that does not justify medical officers of health, who have the duty of making the returns to the Registrar-General, in systematically allowing all notifications to remain in their returns without question, and without subsequent deletion where further investigation would show that a positive swab report, probably on one occasion only, was completely at variance with all the clinical evidence. In the absence of such necessary precautions the Registrar-General's returns will continue more and more to give an erroneous impression of the influence, or lack of it, of immunization on diphtheria the disease.—I am, etc.,

F. LAWRENCE SMITH,
Carshalton. Medical Officer of Health.

SIR,—Dr. G. A. Garrett (Jan. 23, p. 102) discusses the possibility of diphtheria arising during immunization as the result of a negative phase occurring after an injection of A.P.T. For the sake of brevity, I should like to draw his attention to the following points.

1. A negative phase following the injection of any antigen would certainly not last for nearly three weeks, in all probability not more than a few days. It is likely that the importance of the "negative phase" has been overstressed in the past.

2. There is no proof whatever that the case quoted was indeed diphtheria. A positive swab in the absence of definite clinical signs is not a safe criterion of diphtheria, especially as the organism was not typed or other tests for virulence carried out. Moreover, the state of the child's original immunity was unknown. Presumably Dr. Garrett was surprised at the result, as I can only assume the sentence "A negative swab was not anticipated" is an error.

3. Even were the case one of genuine diphtheria of a mild variety this would not be surprising, as the child would be

in exactly the same position to contract the disease as any other non-immunized susceptible child.

4. The statement that the knee-jerks are often absent in early diphtheria is a myth. Even in cases of hypertoxic diphtheria it is rare for the knee-jerks to be lost during the first week. Nor can any reliance be placed on such signs as the height of the temperature, presence or absence of albuminuria, etc., none of which is particularly diagnostic of diphtheria. The decision must be made on the appearance of the throat, and in doubtful cases on a knowledge of the patient's immunity as shown by the Schick test and the results of typing and virulence tests in the laboratory.

5. With regard to Dr. Garrett's comment, first of all there is no ground whatever for assuming that under any circumstances a non-virulent organism can become virulent.

6. If swabbing were resorted to before immunization the presence of virulent bacilli would show that immunization was in fact unnecessary as the patient would surely be Schick-negative, otherwise clinical diphtheria would have developed. In any case the administration of diphtheria antitoxin would not have the slightest effect on the carrier state.

7. Finally, the only advantage which might accrue from giving antitoxin with the immunizing agent would be to procure an immediate and remote immunity by Ramon's method, instead of waiting for active immunity to appear several weeks after the final injection.—I am, etc.,

Plymouth. D. F. JOHNSTONE.

Treatment of Ischaemic Contracture

SIR,—Your annotation (Jan. 23, p. 108) is fully warranted, but it is doubtful whether arteriectomy is primarily necessary. The application of contralateral heat, according to Learmonth's suggestion, is frequently sufficient to release the collateral circulation (*Surgery of Modern Warfare*, first edition, chap. 36, p. 317). I have used this method in three cases of threatened ischaemia, in cases of supracondylar fracture of the humerus in children, and in each of these it has been successful in averting the onset of contracture. (A fourth case where it was unsuccessful was found to have an incomplete lesion of the brachial artery.) The child was put into a bath with the injured limb suspended above water level, and the return of function was almost dramatic. I would earnestly recommend that this measure be tried before operative proceedings are undertaken. —I am, etc.,

Sunderland. W. GRANT WAUGH.

Mitral Systolic Murmurs

SIR,—I read Dr. William Evans's very useful article on mitral systolic murmurs (Jan. 2, p. 8) with great interest and profit. After discussing it with several colleagues, however, I got the impression that it perhaps lends itself to a little confusion. In using the word "irrelevant" one naturally feels that it should mean irrelevant to something or other, and in the terms of the article that something or other is not quite clear. It looks as if Dr. Evans is calling a murmur irrelevant at the mitral valve when it is heard better at some other valve, and not merely when the disease it signifies is in some other valve. The mitral murmur associated with pulmonary stenosis, for instance, is called irrelevant, while that which may be associated with aortic stenosis is not. Would it not be clearer to say outright that "irrelevant" means "irrelevant to disease at the mitral valve." The classification of mitral murmurs would then run as follows: (1) organic—(*a*) relevant, (*b*) irrelevant; (2) non-organic—i.e., innocent.

Accepting Dr. Evans's well-grounded view that mitral incompetence is not established as a pathological entity, the class of relevant mitral murmurs would then be reserved for those signifying mitral stenosis (of which the systolic would, in certain circumstances, be one). In the irrelevant class would fall all those murmurs signifying pathology elsewhere. According to this, the murmurs of pulmonary and aortic stenosis would be classed together, and in the innocent class would fall all those murmurs which could not rightly be identified with pathology anywhere. It would be possible, of course, to divide the irrelevant murmurs into those signifying pathology in the heart (but not in the mitral valve) and those signifying pathology outside the heart. But perhaps this would unnecessarily complicate the classification.

Dr. Evans's main point is that the mind of the clinician should be always correlating what he hears with some definite conception of pathology, or the absence of pathology, and for that reason it would seem desirable that the terms of his classification should relate directly to the site of pathology, if any. This would help to keep his mind on the point, and prevent him from admitting into his nomenclature hazy theories of disease which have never been concretely demonstrated.

Incidentally, Dr. Evans's insistence on the value of the clinical interpretation of heart murmurs will come as a welcome encouragement to those of us who have no gadgets at our command, and will inspire us to recapture some of the clinical acumen which is fast becoming a departed glory.—I am, etc.,

Liverpool. C. H. ROSS CARMICHAEL.

Mass Miniature Radiography Units

SIR,—The letters of Dr. Norman P. Henderson (Jan. 16, p. 81) and Dr. James F. Brailsford (p. 83) raise several important points. While the battle for mass miniature radiography in the Services has been won, the battle for its use in the civil population is only in the preliminary skirmishing phase.

The situation appears to be that about a dozen units have been allotted to cities and another dozen are being distributed among certain counties. To those of us who have worked hard in preparing ourselves for service in this new method and in stimulating interest in local medical and lay circles, the fact that our district is not on the short list for a unit is discouraging. This feeling would quickly be dispelled by a little more information from the Ministry of Health in regard to its policy, with special reference to the expected dates of delivery of the units to the various centres. I believe it to be a fact that local authorities have recently received a memorandum from the Ministry on their proposed scheme for mass radiography, but the radiologists who will (or should) have much to do with the practical work are still much in the dark. Surely it would be helpful if each district could know soon approximately when it might expect to take its part in the national scheme, so that it could set about planning in earnest. No doubt all plans will have to be approved by the Ministry, and this takes time. It is to be hoped that the staffing of units will be delegated to regional authorities who are in the position to pick the best teams. In regions containing two or more large towns, each town, so far as is possible, should be represented by a team member.

Dr. Henderson makes the point that the real province of the "chest physician" is the clinical side of the examination, and suggests that it is a waste of time for him to examine large numbers of normal films. There is much truth in this, and yet the combination of radiologist and physician is better than either alone. That a number of physicians do not act on this principle is a matter for regret. There is a tendency for tuberculosis officers to request hospitals to supply chest films without reports, implying that they are quite competent to interpret them without assistance, and no doubt they are in the majority of cases. But do we not all frequently see films in which there is some point of difficulty or doubt? Do we not then welcome a discussion with a colleague? If, as so often happens, a discussion is impossible, surely the next best thing is to read our colleague's thoughts on paper. Many of the reports will be superfluous, but there will always be a few which contribute some piece of information that would otherwise be missed. I feel strongly that this argument justifies a radiologist's report in every chest case.

One last point in regard to staffing. Uniformity throughout the country in technique and in the classification of reports is essential for success. Cannot this best be achieved by setting up a central school which will train radiographers and organizers of units in a standard method and coach members of viewing teams in approved methods of viewing and classification? To those appalled by such a mechanical concept I would suggest that miniature mass radiography is a mechanical sieve and is not a medium for the art of diagnosis. Uniformity at this stage will not atrophy individual diagnostic skill, which is called for at a later stage. Dr. Brailsford thinks that "it is a defective sieve which will miss the early case." Surely it would be truer to say that it will miss some early cases, but it will find more than it misses. It will not be the fault of miniature

mass radiography if suspicious lesions, unassociated with clinical signs, are allowed to spoil the life of a patient. The assessment of the importance of such a lesion rests with the clinician. The viewer who sees it first in a miniature film is not responsible for its interpretation.

I cannot agree with Dr. Brailsford that the revelation by mass radiography of extensive progressive tuberculous disease is to be regarded as a slur on the medical examination which missed it, for the adequate reason that many such cases have never had a medical examination.—I am, etc.,

Reading. PAUL CAVE.

Ligature of the Innominate Artery

SIR,—In reporting the end-result of a case of ligature of the innominate artery (May, 1932) for subclavian aneurysm in the *Journal* of Oct. 10, 1942 (p. 422), I mentioned 62 recorded cases and said that I was unable to obtain figures for recent years. Subsequently, my attention was called to three additional cases, all of them successful and noteworthy.

The first, a woman aged 65, was operated on by Mr. H. S. Meade of Dublin in November, 1936. The case was published under the title "Compression of an Aneurysm of the Innominate Artery with Fascial Straps" (*Irish J. med. Sci.*). This case was seen in 1942, completely cured. Mr. Charles MacAulay of Dublin operated on a second case—also a woman—in March, 1933. The artery was ligatured. She was shown at the Royal Academy of Medicine in Ireland in 1937, and in 1942 appeared to have made a complete recovery. The third case was reported by Mr. Granville Chapman of Grimsby (*Journal*, July 13, 1939). Mr. Chapman writes that the man is well and alive 14 years after operation.

I am anxious, if you can spare the space, to correct the omission in my original communication by calling attention to these three cases.—I am, etc.,

W. I. DE C. WHEELER.

Artificial Respiration

SIR,—Eve's method has a mechanical but no theoretical superiority over Schäfer's method. In Schäfer's method the operator acts against the force of gravity in the first movement. When he withdraws this force gravity causes the viscera to resume their former position. Only if the body were on a head-downwards incline could the viscera stay put and not resume their former position. The more placid and toneless they are the more vigorous will be the resumption caused by gravity.—I am, etc.,

Birmingham. W. J. BURNS SELKIRK.

Teaching First Aid

SIR,—Should not the teaching of first aid be divided into two distinct categories: (*a*) for the casualty services, founded on the St. John and B.R.C.S. manuals; (*b*) for part-time wardens, rescue parties, Home Guard, W.V.S.—in short, everybody—founded on half a dozen essentials? The reason for this division is that first aid is based on anatomy, physiology, and chemistry, recondite subjects on which the public has little knowledge.

The rules for treatment are, for those under instruction, a matter of memorizing rather than reasoning. This memorizing can only be achieved by constant repetition—a method employed by the casualty services with their weekly or fortnightly drills. This constant repetition is out of the question with wardens, Home Guard, etc., with their many other duties; therefore the rules of treatment have to be reduced to a minimum if they are to be remembered. Six things should be taught: (1) control of haemorrhage by direct pressure; (2) treatment of shock; (3) treatment of broken limbs when no splints are available; (4) how to lift a casualty; and two "don'ts"—(5) don't wash wounds; (6) don't use oil on burns.

A booklet on these lines (10 pp.) was issued by our A.R.P. department eighteen months ago. It improved the first aid of part-time personnel greatly. Should any reader be interested in the views expressed above a copy will be sent if application is made to the A.R.P. Office, Princes Street, Huntingdon.—I am, etc.,

Huntingdon. ARTHUR S. G. BELL, Surg. Commander, R.N. (ret.).

Obituary

SIR STCLAIR THOMSON, M.D., F.R.C.P., F.R.C.S.

Formerly Professor of Laryngology and Physician for Diseases of the Throat and Nose, King's College Hospital

Sir StClair Thomson, whose death occurred on Jan. 29, at the age of 83, achieved fame in every direction in which he bent his energies—as a specialist in diseases of the larynx, as the author of the "Laryngologists' Bible," as a fluent interpreter at international congresses, as a good host, and as a most accomplished speaker. He was the Nestor of British laryngology, almost the sole survivor of the school which had established this specialty on a firm scientific basis.

Although StClair Thomson was born at Londonderry in 1859, his father was an Argyllshire man from the village of Ardrishaig on Loch Gilp. The "StClair" in his son's name was from his maternal grandfather, John Sinclair of the Sound of Mull. When he was 19 StClair Thomson entered as a junior scholar in the medical division of King's College, London. He took the M.R.C.S. and L.S.A. in 1881, and at the M.B. London examination in 1883 he obtained honours in medicine. He then became house-surgeon at King's College Hospital, where his chief was Joseph Lister, who had entered that hospital as a surgeon on the same day that Thomson had entered it as a student. StClair Thomson was never tired of recalling his great master. Fifty-six years after the event, in the rooms of the Medical Society of London in Chandos Street, Cavendish Square, he described the scene in those very rooms in 1883, when he had the honour of bringing together and exhibiting with Lister the first six cases in which a fractured patella had been wired.

StClair Thomson completed his training in Vienna, Paris, and Lausanne, making acquaintance with foreign surgeons and their methods and establishing his knowledge of laryngology. At King's College Hospital at the time he was a student the teaching in this subject was wholly inadequate. He once said that when he gained his degree in medicine he had never seen the vocal cords—or the drum of the ear—in the living person. He also recalled how, when it was necessary to operate for adenoids in the out-patient department in the 'eighties, the struggling child was held by the porter, while the surgeon, whose only instrument was his finger-nail, inserted his finger up behind the palate and scratched out as much of the adenoids as the yelling child would permit.

In Vienna one of his great teachers was Hajek, afterwards professor of laryngology and rhinology at the University there, but then a privat-docent. One of StClair Thomson's possessions which he exhibited proudly half a century later was his postgraduate notes on Hajek's lectures on the anatomy of the nose and throat. It was Hajek who taught him the importance not only of looking at specimens but of handling them and making his own sections. Thomson also worked in Vienna with von Schrötter, Stoerk, and Politzer. In his old age Hajek fell a victim to Nazi persecution and became an exile in this country, and it was the pleasure of StClair Thomson to repay as far as he could the old debt to his teacher by welcoming and acclaiming him in laryngological circles in London.

During his wanderings abroad in his early manhood, which included some experience of practice in Italy and Switzerland, StClair Thomson not only laid the foundation of his knowledge and skill as a laryngologist but also acquired that polished cosmopolitanism which became him so well to the very end of his life. He loved to talk of his time in Vienna, in particular of the long summer of 1893 when the Redehöf (not now to be discovered in the Austrian capital, but commemorated in

the operetta *Lilac Time*) was crowded with students and the life of the University was especially rich and inspiring.

Laryngology in London

In that same year (1893) he returned to London and took the F.R.C.S. He had already taken the London M.D. in 1888, and also the M.D. of Lausanne in 1891. He became physician to the Throat Hospital, Golden Square, and surgeon to the Royal Ear Hospital. In 1901 he was appointed assistant physician for diseases of the throat and nose at his old hospital, King's College, where his department had two beds in the male and two in the female surgical wards, but no special theatre, a room being fitted up for operations. In 1903 he was elected F.R.C.P. In 1905 he became physician in charge of the department, and in 1908 professor of laryngology at King's College. A few years later, in 1913, the hospital migrated from the byways of the Strand to the more open spaces of Denmark Hill. He continued to hold the two appointments until his retirement in 1924 at the age of 65, when he was made consulting surgeon and emeritus professor. His connexion with King's College, so long and distinguished, was further marked by his election to the Fellowship of the College in 1927, and by the unanimous choice of him for the chair of the past and present students' centenary celebrations in 1930.

An adequate estimate of the contributions of StClair Thomson to laryngology cannot even be attempted in the brief space of a memoir. One subject to which he devoted himself in particular was intrinsic cancer of the larynx. In the course of his experience he had seen that condition pass from being regarded as a most dire disease to one of the most amenable from a surgical standpoint, and he himself helped in a marked degree to bring about that change. Laryngofissure, from being an operation with a high mortality, was made safe by improvements in early diagnosis and technique, and this advance was due in large measure to his work. His technique in the operation for malignant disease of the larynx was followed in the schools of laryngology all over the world, as were his methods of after-care, in particular the use of the sitting position for the patient as soon as possible after the operation. In 1927 he exhibited at the Royal Society of Medicine four patients—all medical men—upon whom he had performed the operation of laryngofissure, respectively two and a quarter, three, four and a half, and five and a half years previously, for intrinsic epithelioma, and they were all, at the time he showed them, engaged actively in their practices. He held that if laryngofissure was carried out according to the correct technique it should be free from danger to life and should be followed by an adequate voice and lasting cure. In 1930, with Mr. Lionel Colledge as co-author, he published a book on this subject.

Tuberculosis of the larynx was also one of his great interests, and in a work from his pen in 1924 he described about 2,500 cases which he had seen during thirteen years while holding the appointment of visiting physician for diseases of the throat to the King Edward VII Sanatorium, Midhurst. In 1936 he was presented by the Royal College of Physicians with the Weber-Parkes prize for his work on tuberculosis carried out over a long period. His work as a surgeon was distinguished by the utmost care in examination, courage in undertaking surgical procedures, well-thought-out methods of after-care, and punctilious recording and description. It was he who published the first recorded case in this country, in 1910, of the removal with the aid of endoscopy of a foreign body—a shawl pin—which had been impacted in the secondary bronchus.

His famous textbook, *Diseases of the Nose and Throat*, first appeared in 1911, a second edition in 1916, a third in 1926, and a fourth, in which he was assisted by Mr. V. E. Negus, in 1937. He contributed innumerable papers to the medical journals and societies, and was the European editor of the *Laryngoscope*. His first publication was in 1895—a translation of Ónodi's *Anatomy of the Nasal Cavity*. Among appointments other than those already mentioned which he held at various times were those of surgeon to the throat and ear department of the Dreadnought Hospital, Greenwich, professor of laryngology and otology at the Royal Army Medical College, consulting throat physician to the Actors' and Music Hall Artistes' Associations, and physician to the Royal Italian Opera.

In 1913, when the International Medical Congress was held in London, Sir StClair Thomson—he had received his knighthood in the previous year—was president of the Section of Laryngology, and his command of languages enabled him to welcome each national group of delegates in its own. In 1918 he was president of the Medical Society of London, and in 1924-6 president of the Royal Society of Medicine. It was through his generosity that that society, shortly after he had relinquished the presidency, was granted an Achievement of Arms, and he also presented it with a presidential badge and chain of office, the badge consisting of the shield of the coat of arms encircled by the motto of the society—*Non est vivere sed valere vita.*

StClair Thomson was a corresponding honorary member of most of the laryngological societies of Europe and of the American Laryngological Association. In 1936 the Académie de Médecine of Paris unanimously promoted him, already a Foreign Correspondent, to be a Foreign Associate. Among other noteworthy decorations which he was entitled to wear were the swords of a Commander of the Order of Leopold, the ribbon of an Officer of the Legion of Honour, and the medal of the Reconnaissance Française. On the occasion of the visit of the British Medical Association to Canada in 1930, when he was president of the Section of Laryngology, he received at the Convocation of the University of Manitoba the honorary degree of LL.D. He was president of the same Section at Belfast in 1909 and at the Centenary Meeting in London in 1932. A recognition which perhaps pleased him most of all was the gift from 175 of his fellow laryngologists of a loving cup, which was presented to him in 1926. On that occasion he said that he had had two ambitions—one to till to the best of his ability the small corner of the field of medicine in which he worked; the other to accomplish it in such a way as to keep the esteem and regard, and if possible the affection, of his fellow workers.

Sir StClair Thomson was a widower, his wife, whom he had married in 1900, having died in 1905.

Mr. LIONEL COLLEDGE writes:

The tragic and violent death of Sir StClair Thomson will fill a vast number of friends, pupils, and patients with dismay. It is curious that during the last few years he had made no attempt to conceal from his intimate friends a slightly morbid preoccupation with the end which he felt was approaching, though he might, indeed, have lived for many years, for his health was not bad for a man past 80 and his family is remarkable for its longevity. Yet he could not have had the slightest suspicion of the manner in which his life would be cut short, and it recalls the disaster to the Blue Train in which he was travelling to the Riviera some years ago. Prof. Bérard hastened to the scene of the accident near Lyons and offered accommodation in his clinic. This Sir StClair declined, but soon afterwards found that he was more seriously injured than he had at first supposed. By that time the clinic was full and he had to be content with a place in the *salle des blessés* in the *Hôtel-Dieu*. He gave a vivid description of his sojourn there, which accorded ill with a cat-like love of comfort and elegant living.

The art of living was for him a special study of which he was complete master, and was reflected in the appointments and conduct of his home in Wimpole Street, where he loved to entertain his friends and especially his professional brethren. A recent and sad picture of him, though happily not the last, was sitting in the inner hall of his dismantled house, disposing of the treasures in the home he loved so well and was to occupy no more through the misfortunes of war. His love of hospitality and the social graces did not prevent him from being a worker of more than ordinary industry, and he covered the whole field of his special work in his writings; but he was interested particularly in laryngeal tuberculosis. He had in this a personal interest as one who had in early middle life made a remarkable recovery, which he attributed to the skill and care of his friend Dr. Patrick Watson-Williams, and he always referred to this with gratitude. On this account, too, the Tuberculosis Society always retained his interest and sympathy. His Mitchell lecture on tuberculosis of the larynx and his writing on the same subject for the Medical Research Council based on his work at King Edward VII Sanatorium at Midhurst show how careful a clinician he was and how thoroughly his cases were studied and followed. In this as in other things he preferred quality to quantity. The particular interest in the larynx naturally led

to an intensive study of laryngeal cancer, where the same minute attention to every detail was invoked, and he became the acknowledged clinical authority on the subject. In the operations that he undertook the same qualities—which he had acquired from Lister, who exercised a direct and lifelong influence over him—were displayed, but he never undertook anything which he felt was beyond his powers, and was willing to hand over his cases without the least jealousy or rancour to those upon whom he believed that he could rely. His writings upon these subjects gained him an international reputation, but his enormous influence was exercised far more by his personality, which gained him the friendship of all the leading Continental and American laryngologists, and by his command of the spoken word, and that, too, in several languages. Whether it was after dinner, at a professional meeting, or at a Christmas party at his house, he always spoke with the same felicity and held the attention of his audience with a speech generally pointed with a quotation from his favourite Shakespeare. This combination of professional, oratorical, literary, and social ability would have led him to distinction in any line of life, and had he followed some other path he would have held first place at Canterbury, Westminster, or the Mansion House, and it is easy to picture him as an Ambassador.

That he had countless friends followed naturally, but he used to say that those friendships were the deepest and most enduring which came to him in the course of his work, and so by those who worked with him and reaped the benefit of his courteous instruction, his kindness, and his knowledge he will be remembered as the friend of laryngologists.

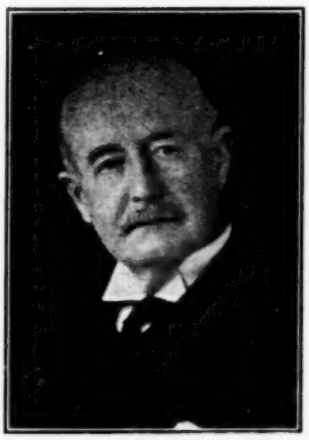
Mr. MCADAM ECCLES writes:

Sir StClair Thomson from the very first took the greatest interest in the University of London Medical Graduates Society, for it was in his house in Wimpole Street it was born on Oct. 31, 1927. He became its first president, and presided at the inaugural dinner on Feb. 13, 1928, at the Langham Hotel. The society has gone on from strength to strength because of his wise guidance and sound judgment, and has now a membership of over 600. His genial presence, graceful oratory, and loyal friendship will be greatly missed by all.

SIR ROBERT ARMSTRONG-JONES, C.B.E., M.D., F.R.C.P., F.R.C.S.

We regret to announce the death on Jan. 31 at the age of 85 of Sir Robert Armstrong-Jones, consulting physician in psychological medicine to St. Bartholomew's Hospital.

Robert Armstrong-Jones was born in 1859 at Ynyscynhaiarn, Caernarvonshire, the son of the Rev. Thomas Jones, a Congregationalist minister who had formerly belonged to the Church of England. Robert Armstrong-Jones was one of a family of ten, and he used to recall how as a young boy he walked three and a half miles daily to and from Portmadoc Grammar School, where he received a prize for being the most regular and constant attendant during a severe winter quarter in 1871. When he was 14 he moved to the University College of Wales at Aberystwyth, and from there to Grove Park School, Wrexham. He took the London Matriculation in 1875, and after having served six months' apprenticeship to a Dr. Robert Roberts at Portmadoc he entered St. Bartholomew's Hospital. At Bart's he was prosector in anatomy and gained the Wix Essay and Hichens Prize. He qualified L.S.A. in 1880, took the London M.B. in the same year, and proceeded to the M.D. in 1883. He took the F.R.C.S. in 1885, and in 1900 the M.R.C.P., being elected Fellow in 1907. In 1920 he was given the Honorary D.Sc. of Wales University.



[*Press Portrait Bureau*

Leaving Bart's in 1880, Armstrong-Jones was for two years junior medical officer to the Royal Earlswood Institution, and then for six years at the Colney Hatch Asylum. In 1888 he returned to Earlswood as medical superintendent. After six years' service in this capacity he was appointed in 1893 the first medical superintendent to the first new asylum of the London County Council at Claybury; at the same time Dr. (later Sir) Frederick Mott was appointed pathologist. Jones's energy, enthusiasm, tact, and administrative ability made the renown of Claybury as an institution spread far beyond the area controlled by the L.C.C. It was the first asylum under municipal control to receive private paying patients, and it is interesting to recall these words from the first annual report: "It is hoped that the success of this experiment may encourage the Council to provide further for a class of patients above the pauper class but who can ill afford to bear the cost of private asylum, and who may find comparative quiet and comfort—perhaps even a touch of 'home'—in such an institution as the Claybury mansion-house." Armstrong-Jones organized the teaching of mental nurses and instituted a special training course for them, and his work on behalf of mental nursing was recognized by the conferment upon him of Knight of Grace by the Order of St. John of Jerusalem. Whilst Armstrong-Jones was superintendent, Frederick Mott was at the same time working in the pathological laboratory and making his fundamental and well-known contributions to the pathology of insanity. Armstrong-Jones was for several years honorary general secretary to the Medico-Psychological Association, and later became its president. With F. E. Batten he was joint secretary of the Psychological Section of the International Congress on School Hygiene which met in London in 1907.

After nearly twenty-five years of arduous service at Claybury, he felt compelled by the stress and strain of administrative and medical work to resign his appointment in 1916; in the following year he received the honour of knighthood. After a short time of rest Armstrong-Jones offered his services to the War Office and was appointed first consulting physician in mental diseases to the London Command. He was also consulting physician to the American Red Cross Hospital at Alford House. His war services were recognized by the honour of C.B.E. (Military). After the war he was appointed to be one of the three Lord Chancellor's Visitors in Lunacy, an office he held for ten years. Later he was nominated Justice of the Peace and Deputy Lieutenant for the Counties of London and Caernarvon, and in 1929 he was High Sheriff of Caernarvonshire. Armstrong-Jones lectured in mental diseases both at St. Bartholomew's Hospital and at the Westminster Hospital, published a *Textbook of Mental and Sick Nursing*, and contributed articles on insanity to *Quain's Dictionary of Medicine* and to *Allbutt's System of Medicine*. He contributed articles to the general medical press and often vigorously expounded his views in the correspondence columns of the *Times*. In 1910 he was witness for the British Medical Association before the Royal Commission for Divorce and Matrimonial Causes, and served on a special Committee to Consider Spiritual Healing appointed by the Archbishop of Canterbury in 1920. In 1929 he delivered the Henderson Trust Lecture in the University of Edinburgh, the title of his lecture being "The Growth of the Mind." From 1917 to 1927 he was Gresham Professor of Physic.

Sir Robert Armstrong-Jones was actively interested in the affairs of the B.M.A.: he was secretary of the Section of Psychology at the Meeting at Newcastle-upon-Tyne in 1893, and president at the Swansea Meeting in 1903; in 1920 and in 1921 he was a member of the executive committee of the Kensington Division.

He married in 1893 Margaret Elizabeth, the elder daughter of Sir Owen Roberts, D.C.L.; they had one son and two daughters.

We regret to announce the death of Dr. ROBERT CRAIK of Ealing at the age of 77. Dr. Craik received his medical education at Glasgow University, qualifying M.B., C.M. there in 1886, and obtaining his M.D. in 1893. For many years he was medical superintendent of Conisborough Fever Hospital, Yorks, before going to Ealing in 1912. From time to time he contributed to the correspondence columns of the *Journal* on subjects in which he took a special interest, including the blood in lead poisoning and the blood picture 35 years after splenectomy. Dr. Craik's modest and retiring disposition kept him too much in the background, but his real worth was appreciated by all who came in contact with him. He had been a member of the B.M.A. for 20 years.

The Services

CASUALTIES IN THE MEDICAL SERVICES

We have recently received from Australia the annual report for 1942 of the Victorian Branch of the B.M.A. The following medical officers are included in the Roll of Honour printed in the report:

Died on Service.—Major E. Bailhache, Major J. F. Chambers, Lieut.-Col. E. L. Cooper, Capt. J. C. R. Joyce, Capt. G. L. Lindon, Major Z. Schwartz, Capt. D. J. Shale, and Capt. S. I. Weir, all A.A.M.C., and Fl. Lieut. S. Thomson, R.A.A.F. A brief obituary notice of Capt. Weir was published in the *Journal* of Sept. 6, 1941 (p. 361).

Major ERIC BAILHACHE was educated at the University of Melbourne, where he graduated M.B., B.S. in 1924. He also took the M.R.C.O.G. in 1937. He died of wounds on Feb. 26, 1941. He had been a member of the B.M.A. since 1924.

Major JOHN FERGUSON CHAMBERS graduated M.B., B.S. of the University of Melbourne in 1917, proceeding M.D. in 1925. He also took the M.R.C.P. in 1922. He was honorary physician with charge of in-patients at the Alfred Hospital, Melbourne, and honorary consulting physician to the Austin Hospital, Melbourne. He was killed in action in April, 1941. He had been a member of the B.M.A. since 1918.

Lieut.-Col. ERIC LEONARD COOPER graduated M.B., B.S. of the University of Melbourne in 1925, proceeding M.D. two years later. He was tutor in medicine at the Ormond College of the University of Melbourne and honorary physician to in-patients at St. Vincent's Hospital, Melbourne. He contributed articles to the *Medical Journal of Australia* on Hodgkin's disease (1934) and on athletics and the heart (1937). He joined the B.M.A. in 1925.

Capt. JOHN COLIN RAMSAY JOYCE took the M.B., B.S. of the University of Melbourne in 1926 and joined the B.M.A. in the same year.

Capt. GEORGE LEONARD LINDON graduated M.B., B.S. of the University of Melbourne in 1940 and joined the B.M.A. in the following year.

Major ZELMAN SCHWARTZ graduated M.B., B.S. of the University of Melbourne in 1920 and took the F.R.C.S.Ed. in 1924. He joined the B.M.A. in 1920.

Capt. DONALD JAMES SHALE took the M.B., B.S. of the University of Melbourne in 1938.

Fl. Lieut. STUART THOMSON graduated M.B., B.S. of the University of Melbourne in 1937. He entered the R.A.A.F. soon after the outbreak of war and was killed in a flying accident. He joined the B.M.A. in 1940.

Believed to be Prisoners of War.—Major J. F. Akeroyd, Capt. B. H. Anderson, Major H. L. Andrews, Capt. V. Brand, Capt. V. G. Bristow, Major K. B. Burnside, Capt. J. F. J. Cade, Capt. F. J. Cahill, Major I. T. Cameron, Capt. J. P. Catchlove, Lieut.-Col. A. E. Coates, Major T. P. Crankshaw, Col. A. P. Derham, Major E. E. Dunlop, Lieut.-Col. N. M. Eadie, Major H. H. Eddey, Capt. J. L. Frew, Major R. B. Maynard, Capt. P. N. O'Donnell, Major R. G. Orr, Lieut.-Col. C. H. Osborn, Major H. A. Phillips, Col. D. C. Pigdon, Major J. J. Searby, Capt. H. N. Silverman, Lieut.-Col. H. F. Summons, Capt. H. F. Tucker, Capt. F. R. Vincent, Major H. A. W. Watson, Col. E. R. White, Lieut.-Col. J. G. G. White, Capt. M. F. A. Woodruff, all A.A.M.C.

Missing.—Capt. C. S. Donald, A.A.M.C., Surg. Cmdr. J. R. Hasker, R.A.N., Surg. Lieut.-Cmdr. F. H. Genge, R.A.N., Surg. Lieut. W. J. McLaren-Robinson, R.A.N., Capt. J. F. Park, A.A.M.C., Surg. Lieut.-Cmdr. E. M. Tymms, R.A.N.

Universities and Colleges

UNIVERSITY OF CAMBRIDGE

At a Congregation held on Jan. 22, 1943, the following medical degrees were conferred by proxy:

M.D.—H. H. Bayley, C. W. C. Karran.

M.B., B.CHIR.—A. E. Howarth, A. D. Barlow, J. F. B. Carter, D. S. G. Genge, R. N. Jones, T. G. E. Loosemore, R. D. Slack, H. C. Still, J. K. F. Mason, W. N. Coombes, R. K. Reid, J. H. Simpson, R. N. Ticehurst, W. H. Trethowan, O. B. Appleyard, N. A. Campbell, J. A. H. Collyns, R. V. Peters, W. Rogers, M. C. Connell, P. H. N. Matthews, J. F. Neil, D. W. H. Griffiths, M. C. Mundle, B. P. Webber, J. H. C. Phillips, A. P. H. Randle, W. A. dos Santos, A. G. Harrold, A. G. Richards, P. F. Egrly, F. W. Blackley, E. F. Carr, W. K. Douglas, H. L. Pierce, H. R. Jolly, M. G. P. Stoker, B. C. Welshman. *In person:* H. W. A. Baron, J. V. T. Gostling.

The dissertation by M. J. Clow, M.B., B.Chir., has been approved for the degree of M.D. in absence.

UNIVERSITY OF LONDON

A course of lectures and clinical instruction on mental deficiency and allied conditions, arranged by the University Extension and Tutorial Classes Council in co-operation with the Central Association for Mental Welfare, will be given from Monday, May 10, to Friday, May 21, at the London School of Hygiene and Tropical Medicine, Keppel Street, W.C. It is intended for medical practitioners, more especially those who are engaged as school medical officers, certifying officers to local authorities under the Mental Deficiency Acts, or as medical officers of institutions. Detailed time-tables will be sent not later than May 3 to each person proposing to attend the course, and applications should be sent in by March 20 as the course can be held only if sufficient entries are received. The University will grant certificates of attendance to those students who attend regularly taking both theoretical and practical work. The registration fee, to be paid at the time of application, is 10s. 6d., and the fee for the course is £5 15s. 6d. All communications should be addressed to Miss Evelyn Fox, C.B.E., c/o University Extension Department, University of London, 24, Buckingham Palace Road, London, S.W.1.

UNIVERSITY OF EDINBURGH

At a graduation ceremonial on Jan. 21 the following medical degrees and diplomas were conferred:

M.D.—*J. W. Buchanan, Surg. Lieut., R.N.V.R., *R. F. Dawson, Squad. Ldr., R.A.F.V.R., *O S. Gibbs, *N. S. Gordon, Flying Officer, R.A.F.V.R., *W. Limont, Surg. Lieut., R.N.V.R., J. A. F. Roberts (highly commended for thesis).

M.B., CH.B.—P. H. L. Barker, G. T. Bedford, G. V. R. Born, M. A. Caldwell-Nichols, E. M. Callander, K. M. Chalmers, T. B. Chetty, D. H. Clark, *W. A. L. Collier, D. J. C. Cunningham, Helen W. Currie, K. J. R. Cuthbert, D. A. Dixon, W. Edgar, G. M. Falconer, E. C. Fear, Margaret M. Fleming, J. P. W. Grant, D. R. Gunn, K. Gunstensen, W. J. Howrie, D. W. Huish, W. H. Hunter, R. B. Huston, B. C. Jeffrey, D. R. Kerr, W. A. E. Kerr, A. D. M. King, R. P. T. King, W. T. Lesslie, D. W. Livingstone, D. G. M'Connell, A. C. Macdonald, Evelyn E. M'Guinness, Annie H. Mackay, L. M'Lachlan, A. S. M'Lean, C. W. Magill, R. M. Marquis, A. R. Milson, N. R. K. Mitchell, I. Payne-James, G. Reid, P. M. Roemmele, E. B. Ross, W. T. Scott, C. W. Shearer, I. B. Sim, I. Singh, A. S. Smith, H. T. G. Strawbridge (with honours), C. R. Strother-Stewart, R. S. Sunderland, A. R. Sutherland, J. A. Tulloch, G. A. S. Wallace, J. L. Watson, A. W. Williams, Lena W. Williams, H. D. Wilson, A. F. Young.

DIPLOMA IN PUBLIC HEALTH.—*Winefride M. Hamilton, *Doreen G. Warnock.

DIPLOMA IN MEDICAL RADIOLOGY.—E. Tiscenco.

* *In absentia.*

POLISH SCHOOL OF MEDICINE IN EDINBURGH

M.B., CH.B.—H. Drobinski, T. W. Drozdowski, F. J. Kuciel, W. S. Kulesza, T. Markiewicz, K. J. Parkita, S. J. Rydlewski, H. Sulinska.

ROYAL FACULTY OF PHYSICIANS AND SURGEONS OF GLASGOW

Surg. Rear-Admiral Cecil P. G. Wakeley will deliver a lecture, under the auspices of the Dr. John Burns Fund, on "The Surgical Aspects of Blast" in the hall of the Faculty (242, St. Vincent Street, Glasgow) on Wednesday, Feb. 10, at 4 p.m.

Medical Notes in Parliament

The National Loaf

Mr. MABANE stated on Jan. 20 that, apart from yeast, salt, and the various improvers which are the recognized adjuncts of bread baking, the permitted ingredients of national flour for making the present national loaf are wheat flour of 85% extraction, imported white flour, oats products, barley, rye, milk powder, and calcium, in the proportions authorized. In addition the baker may use a proportion of white flour as permitted in Article 20 of the Flour Order, 1943, and potatoes and potato flour as permitted in the Bread (Control and Maximum Prices) Order, 1943. The composition of national flour and consequently of national bread is not, and will not, be standardized over the whole country. The proportion of diluents does not at present in general exceed 5%. There was no present intention of rationing bread.

Milk Pasteurization

Mr. DAVID ADAMS asked on Jan. 20 whether, with a view to destroying pathogenic organisms in non-pasteurized milk, it was now intended that all milk for human consumption should be scientifically pasteurized. Mr. MABANE replied that the appropriate and practical steps which could be taken to secure the elimination of active tubercle organisms from liquid milk were at present under consideration with the Agricultural and Health Departments and other interests.

Medical Certificates

Mr. ERNEST BROWN said on Jan. 21 that he was aware of the complaints of doctors that they were overwhelmed with the signing of certificates. In many cases the granting of some exemption or privilege must depend on the physical condition of the applicant. The only suitable evidence of this was a medical certificate. He was grateful to the medical profession for the assistance they had given. He had been consulted both by the other Government Departments concerned and by the medical profession. He was always ready to discuss proposals for restricting, and if possible reducing, the number of purposes for which medical certificates were required.

Treatment of Syphilis under Regulation 33B

On Jan. 26 Dr. MORGAN asked the Attorney-General whether, in view of the risks inherent in the modern treatment of syphilis by drugs containing arsenic, he had considered the probability of action for damages or legal suits against the special practitioners in connexion with their work under the compulsory treatment powers of Regulation 33B, and the position if a breadwinner died or could prove permanent disability as the result of enforced treatment. Mr. ERNEST BROWN, who replied, said the construction of the Regulation was a matter for the courts and not for him to determine, but he did not think that a patient who was treated under the Regulation was in a different position as regards claims against the practitioner than a patient treated by a practitioner who had been consulted in the usual way.

Prisoners of War in Hong Kong

On Jan. 26 Mr. A. HENDERSON informed Sir William Davison that it was believed that the majority of the prisoners of war had been transferred from Hong Kong, but it was probable that some 2,000 British and Canadian as well as some Indian prisoners remained there. The International Red Cross Committee delegate reported favourably on the Hong Kong camps in Oct., 1942, but later information indicated a deterioration in the rations and considerable outbreaks of disease during last summer. It was understood that prisoners of war admitted to hospital received adequate nursing, but that medical treatment was hampered by a serious shortage of drugs and other medical requisites.

EPIDEMIOLOGICAL NOTES

Discussion of Table

In *England and Wales* there were during the week further falls in the incidence of measles 1,830, and diphtheria 77; the notifications of whooping-cough and acute pneumonia continued on the upgrade—by 202 and 103 respectively. An increase of 149 cases of scarlet fever was recorded; the notifications of dysentery were down by 23.

Scarlet fever tended to be more prevalent in most counties, especially in Middlesex 28, Yorks West Riding 27, London 24, and Glamorganshire 33 cases more. Lancashire, with 31 fewer notifications than in the preceding week, provided the only exception of note to the general trend.

The returns from the counties for diphtheria showed only slight changes from the totals of the previous week. The only substantial variations were a rise of 21 in Lancashire and a fall of 46 in Yorks West Riding.

The total notifications of whooping-cough were higher than in the previous week. The largest increases were in Lancashire 45, Staffordshire 28, London 26, and Somersetshire 20.

Although measles was down by 13% for the whole country, notifications rose in several counties, especially in Hampshire 70, Yorks North Riding 61, and Kent 51. The greatest decreases were those of Yorks West Riding 433, Durham 296, Lancashire 222, Lincolnshire 166, Derbyshire 142, and Staffordshire 106.

Half the total notifications of dysentery were from three counties: London 18, Essex 15, Lancashire 14. The epidemic of dysentery in Middlesbrough C.B. is apparently subsiding, only 4 cases being reported during the week; there are only 14 persons under hospital treatment.

In *Scotland* measles was down by 148 cases; notifications of acute primary pneumonia were up by 60 and of whooping-cough by 48. A suspected case of small-pox in a 3-year-old girl has been reported from Bridge of Allan. She has been removed to an Edinburgh institution after a period of observation in a Stirling hospital.

Typhus in Germany

A League of Nations report states that a large increase of typhus has occurred among the civilian population in Germany during the war. In 1939 no civilian case occurred, in 1940 there were 6, and in 1941 the total rose to 395. During the first five months of 1942 there were 3,674 cases. Germany ceased to issue returns in September.

Returns for the Week Ending January 23

The notifications of infectious diseases in England and Wales during the week included: scarlet fever 2,203, whooping-cough 1,841, diphtheria 998, measles 11,827, acute pneumonia 1,404, cerebrospinal fever 92, dysentery 113, paratyphoid fever 1, typhoid 7. In the large towns 100 deaths were ascribed to influenza.

No. 2

INFECTIOUS DISEASES AND VITAL STATISTICS

We print below a summary of Infectious Diseases and Vital Statistics in the British Isles during the week ended Jan. 16.

Figures of Principal Notifiable Diseases for the week and those for the corresponding week last year, for: (a) England and Wales (London included). (b) London (administrative county). (c) Scotland. (d) Eire. (e) Northern Ireland.

Figures of Births and Deaths, and of Deaths recorded under each infectious disease, are for: (a) The 126 great towns in England and Wales (including London). (b) London (administrative county). (c) The 16 principal towns in Scotland. (d) The 13 principal towns in Eire. (e) The 10 principal towns in Northern Ireland.

A dash — denotes no cases; a blank space denotes disease not notifiable or no return available.

Disease	1943 (a)	(b)	(c)	(d)	(e)	1942 (Corresponding Week) (a)	(b)	(c)	(d)	(e)
Cerebrospinal fever	96	8	24	4	3	177	13	33	4	7
Deaths		2	1				1	1		
Diphtheria	764	45	231	97	31	1,013	38	249	29	37
Deaths	27	—	2	6	1	33	1	8	2	1
Dysentery	96	18	31	—	—	128	7	32	—	—
Deaths			—	—	—			—	—	—
Encephalitis lethargica, acute	2	—	—	—	—	—	—	—	1	1
Deaths		—	—				—	—		
Erysipelas		—	55	11	3		—	55	5	2
Deaths		—	—				—	—		
Infective enteritis or diarrhoea under 2 years				23						
Deaths	41	3	11	17	4	33	2	9	7	—
Measles	12,323	630	377	18	34	896	77	81	65	5
Deaths	16	—	3	—	1	1	—	—	—	—
Ophthalmia neonatorum	87	2	18		—	61	1	20		—
Deaths										
Paratyphoid fever	5	—	—	—		8	—	1	—	
Deaths	1	—	—	—	1	—	—	—	—	—
Pneumonia, influenzal*	1,194	73	12	9	3	1,028	72	19	3	11
Deaths (from influenza)	89	4	2	1	—	62	15	6	—	3
Pneumonia, primary			276	13				363	13	
Deaths		—		12	8		54		16	14
Polio-encephalitis, acute	—	—				—	—			
Deaths										
Poliomyelitis, acute	8	1	—	8	3	6	—	1	—	—
Deaths			—					—		
Puerperal fever	—	5	15	—	—	3	3	4	1	—
Deaths		—					—			
Puerperal pyrexia	159	10	15		3	159	11	16		2
Deaths										
Relapsing fever	—	—			—	—	—			—
Deaths										
Scarlet fever	1,957	158	339	57	52	1,047	32	223	38	35
Deaths	—	—	1	—	—	2	—	—	—	—
Small-pox	—	—	—		—	—	—	—		—
Deaths	—	—	—	—	—	—	—	—	—	—
Typhoid fever†	4	—	1	3	—	24	—	1	6	—
Deaths	—	—	—	—	—	1	—			—
Typhus fever	—	—	—	—	—	—	—	—	—	—
Deaths			—	—	—			—	—	—
Whooping-cough	1,504	112	124	45	8	1,601	177	69	22	3
Deaths	10	—	2	3	1	16	6	3	1	—
Deaths (0–1 year)	454	43	74	50	19	398	48	78	28	22
Infant mortality rate (per 1,000 live births)										
Deaths (excluding stillbirths)	6,003	857	696	261	150	5,557	820	721	245	157
Annual death rate (per 1,000 persons living)			15·7	17·2	‡			16·2	16·3	‡
Live births	6,519	804	910	393	286	4,942	494	826	295	206
Annual rate per 1,000 persons living			18·6	25·8	‡			17·1	19·6	‡
Stillbirths	238	28	43			229	12	44		
Rate per 1,000 total births (including stillborn)			45					51		

* Includes primary form for England and Wales, London (administrative county), and Northern Ireland.

† Includes paratyphoid A and B for Northern Ireland.

‡ Owing to evacuation schemes and other movements of population, birth and death rates for Northern Ireland are no longer available.

Medical News

A paper on "Measures for the Control of Malaria in India" will be read by Sir S. Rickard Christophers, F.R.S., at the Royal Society of Arts, John Adam Street, Adelphi, W.C., to-day (Friday, Feb. 5) at 1.45 p.m.

A week-end course for medical practitioners on factory medical services and industrial diseases will be held at the London School of Hygiene on Feb. 27 and 28. The proceedings will be opened by Sir Wilfred Garrett, H.M. Chief Inspector of Factories, at 2 p.m. on Feb. 27; at 2.15 p.m. Dr. A. J. Amor will speak on "Industrial Diseases of the Lungs"; and at 4.15 p.m. Dr. W. D. Jenkins will give an address on "Skin Lesions arising from Coal Distillation." The programme for Feb. 28 is as follows: 10.15 a.m., Dr. M. W. Goldblatt, "The Investigation of Toxic Hazards"; 11.45 a.m., Dr. H. B. Trumper, "A Method of Assessment of Physical Requirements for Processes, with Reference to Routine Medical Examinations"; 2.15 p.m., Dr. A. H. Bennett, "Industrial Fracture"; 3.45 p.m., Dr. A. R. Martin, "Recent Work on Antiseptics." The fee for the course is £1 1s. (plus 2s. 6d. if lunch is required), and applications should reach the secretary of the school (Keppel Street, London, W.C.1) by Feb. 22.

The morning session of a meeting of the Royal Sanitary Institute, to be held at Leicester on Feb. 20, will be devoted to two papers, one on tuberculosis in wartime by Dr. J. C. H. Mackenzie, and the other on the milk supply of the future by Mr. W. W. Baum and Mr. F. G. McHugh, respectively chief sanitary inspectors of the county and the city of Leicester. Visits arranged for the afternoon include an inspection of a high-temperature-short-time pasteurization plant. Further information may be obtained from the hon. local secretary, Dr. E. K. Macdonald, M.O.H., Grey Friars, Leicester.

The British Film Institute is arranging a week-end conference from April 2 to 5 on the film in national life. The conference will be held at the University College of the South-West, Exeter, and will be concerned especially with the influence of the film on society, with particular reference to children and adolescents. It is hoped to show examples of apparatus used in connexion with the employment of optical aids for instructional purposes. It is understood that the fees for the week-end should not exceed 3½ guineas. Further inquiries should be addressed to the British Film Institute, 4, Great Russell Street, London, W.C.1.

Dr. Henry Warner Collins (Inner Temple) and Dr. John Walter Hulme (Middle Temple) were called to the Bar on Jan. 26.

A Swiss Paracelsus Society has recently been founded under the presidency of Prof. Linus Birchler. Its organ will be entitled *Nova Acta Paracelsica*.

According to an account in a Berlin newspaper last December, a man in Frankfurt was fined 50 marks for summoning a doctor to a patient without sufficient reason.

Letters, Notes, and Answers

All communications in regard to editorial business should be addressed to THE EDITOR, BRITISH MEDICAL JOURNAL, B.M.A. HOUSE, TAVISTOCK SQUARE, LONDON, W.C.1.

ORIGINAL ARTICLES AND LETTERS forwarded for publication are understood to be offered to the *British Medical Journal* alone unless the contrary be stated.

Authors desiring REPRINTS should communicate with the Secretary of the Journal Board, B.M.A. House, Tavistock Square, W.C.1, on receipt of proofs. Authors over-seas should indicate on MSS. if reprints are required, as proofs are not sent abroad.

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TELEPHONE NO.—B.M.A. and *B.M.J.*: EUSTON 2111.

TELEGRAPHIC ADDRESSES.—EDITOR, *Aitiology Westcent, London;* SECRETARY, *Medisecra Westcent, London.*

B.M.A. SCOTTISH OFFICE: 7, Drumsheugh Gardens, Edinburgh.

QUERIES AND ANSWERS

Income Tax

Inquiries by Inspector

"NOSEY" exercised "the very strictest economy as to living expenses" in the first year of the war and lent all the money possible for the war effort. The inspector of taxes, noticing that the professional account showed smaller drawings than usual for living expenses, asked for particulars of the private expenditure on food, clothing, holidays, etc. This information was given, but had the inspector a right to ask for it?

*** The legal rights of an inspector of taxes are very limited, and such inquiries are made not by virtue of any authority *he* has, but because the assessing commissioners may wish to know, and such particulars can normally be required on appeal. In the circumstances referred to above the inspector's action seems to suggest too much zeal on his part.

Retired Practitioner—New Appointment—Residential Expenses

"X" is a retired practitioner living at B. He contemplates accepting a whole-time appointment at C. He would (for personal reasons) have to maintain his establishment at B. What can he claim for the expense of maintaining himself at C?

*** The income tax Acts do not provide for any such allowance, and "X" would be liable to account for tax on his full salary. A possible remedy would be for the appointment to be on a "living-in" basis, the result being to reduce the monetary emoluments and consequently the income tax liability.

LETTERS, NOTES, ETC.

Nasal Defence

Dr. OCTAVIA LEWIN (London) writes: The nose is almost universally described as the organ of smell and classified among the special senses. Should not rhinology rather be dealt with as a special branch of physiology and recognized as the first in importance of Nature's many defence measures? The Ministry of Health has published alarming statistics telling of the ravages of the common cold and the losses that the war machine is suffering in consequence. The grave increase in the incidence of tuberculosis may well be ascribed to failure of the germicidal powers of the nasal apparatus. The nasal toilet, which alone can keep the nose fighting fit, is handicapped through lack of attention during the school years, and difficulties have been increased since coupons are called for when hankies are needed, as they form an essential part of our personal equipment in a civilized community.

"British Pharmacopoeia" Amendments

The General Medical Council has amended the *British Pharmacopoeia, 1932,* as follows:

Extractum Cascarae Sagradae Siccum.—When this is prescribed or demanded an extract prepared according to the following formula may be dispensed or supplied: Mix 900 grammes of cascara sagrada, in coarse powder, with 4,000 millilitres of boiling water, and macerate the mixture during three hours. Then transfer it to a percolator, allow it to drain, and exhaust it by percolation, using boiling water as the menstruum and collecting about 5,000 millilitres of percolate. Evaporate the percolate to dryness, reduce the extract to a fine powder, and add sufficient starch, dried at 100° C., to make the product weigh 300 grammes. Mix the powders thoroughly and pass the extract through a fine sieve.

Ferri Sulphas Exsiccatus.—The content of $FeSO_4$ is changed from "not less than 80%" to "not less than 77%."

Paraffinum Molle Album.—When white soft paraffin is prescribed, yellow soft paraffin may be dispensed, and may be used in place of white soft paraffin in making the preparations of the *B.P.*

The Chemist's Shop in Denmark

Dr. K. E. BARLOW (Coventry) writes: It is too little known that there are countries which have dealt radically and adequately with the questions of self-medication. Having been fined by the Coventry panel committee for excessive prescribing, I inquired into the arrangements in Copenhagen when I was there in 1939. Danish chemists are forbidden to sell medicine without a prescription from a doctor. Manufacturing chemists have not only to disclose their formulas but also their costs. A medical board only allows the manufacture of proprietary remedies when the sale price bears a relation to cost which is thought to be just. It is noteworthy that the manufacture of amidopyrine has been prohibited in Denmark—that in consequence the incidence of agranulocytosis is negligible. The appearance of the chemist's shop is characteristic of the arrangements which govern its business. It contains a full pharmacopoeial range in neat bottles and little else. The chemist must live on the premises and must get up at night when required to dispense urgent medicines. Finally, excessive prescribing is checked by calling upon the patient to pay a small proportion of the cost of drugs—a cost which the Government ensures is reasonable. In Denmark doctors are never fined for using the therapeutic resources which are available to them.